CORELATION OF CLINICAL AND IMAGING STUDIES IN ASSESSING THE SEVERITY IN ACUTE PANCREATITIS

Dissertation submitted to
The Tamil Nadu Dr. M.G.R.
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With fulfilment of the regulations
for the award of Degree

M.S. GENERAL SURGERY
BRANCH – I

DEPARTMENT OF SURGERY
K.A.P.V. GOVT. MEDICAL COLLEGE,
TRICHY.

APRIL 2014

CERTIFICATE

This is to certify that this dissertation titled "**CORELATION OF CLINICAL AND IMAGING STUDIES IN ASSESSING THE SEVERITY IN ACUTE PANCREATITIS**" is a bonafide work of **DR.SHANKAR M. R.**., Post Graduate in M.S. General Surgery, Department of General Surgery, K.A.P.V. Government Medical College, Trichy and has been prepared by him under our guidance. This has been submitted in partial fulfilment of regulations of The Tamil Nadu Dr. M.G.R. Medical University, Chennai -32 for the award of M.S. Degree in General Surgery (Branch- I)

Prof. Dr. A.Kanaka Sundaram, M.S.,
Professor and Head of Department,
Department of Surgery,
K.A.P.V. Govt. Medical College,
Trichy.

Prof. Dr. P.Rajagopal.M.S.
Professor and Unit Chief,
Department of Surgery,
K.A.P.V. Govt. Medical College,
Trichy.

Prof.Dr. Vallinayagam,M.D.,(FM)
Dean,
K.A.P.V. Government Medical College,
Trichy.

DECLARATION

*I **Dr. SHANKAR. M.R**, solemnly declare that this dissertation titled, **"CORELATION OF CLINICAL AND IMAGING STUDIES IN ASSESSING THE SEVERITY IN ACUTE PANCREATITIS"** is a bonafide work done by me at K.A.P.V. Government Medical College, during 2011-2014 under the guidance and supervision of my Unit Chief **Professor. Dr. P. Raja Gopal. M.S.***

The dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University, towards the partial fulfillment of requirement for the award of M.S. Degree (Branch – I) in General Surgery.

Place: Trichy

Date : **Dr. SHANKAR.M.R.**

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This is to certify that the project work titled **Correlation of clinical and imaging studies in Assessing the severity in acute pancreatitis** proposed by **Dr. Shankar M.R**. Part of fulfillment of M.D/M.S course in the subject of **Surgery** for the year **2011-2014** by The Tamilnadu Dr.MGR Medical University has been cleared by the ethical committee.

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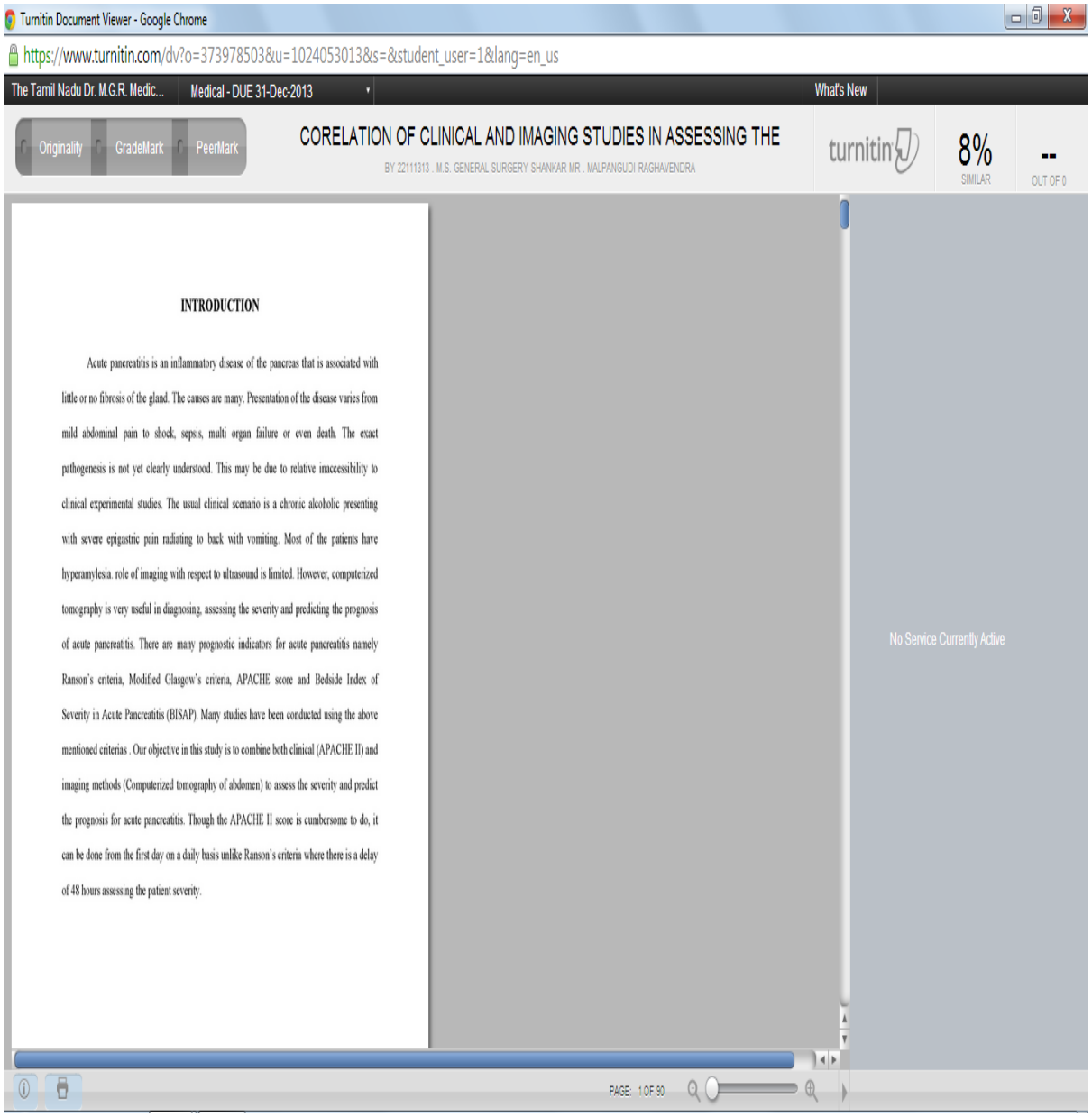
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INTRODUCTION
Acute pancreatitis is an inflammatory disease of the pancreas that is associated with little or no fibrosis of the gland. The causes are many. Presentation of the disease varies from mild abdominal pain to shock, sepsis, multi organ failure or even death. The exact pathogenesis is not yet clearly understood. This may be due to relative inaccessibility to clinical experimental studies. The usual clinical scenario is a chronic alcoholic presenting with severe epigastric pain radiating to back with vomiting. Most of the patients have hyperamylesia. role of imaging with respect to ultrasound is limited. However, computerized tomography is very useful in diagnosing, assessing the severity and predicting the prognosis of acute pancreatitis. There are many prognostic indicators for acute pancreatitis namely Ranson's criteria, Modified Glasgow's criteria, APACHE score and Bedside Index of Severity in Acute Pancreatitis (BISAP). Many studies have been conducted using the above mentioned criterias . Our objective in this study is to combine both clinical (APACHE II) and imaging methods (Computerized tomography of abdomen) to assess the severity and predict the prognosis for acute pancreatitis. Though the APACHE II score is cumbersome to do, it can be done from the first day on a daily basis unlike Ranson's criteria where there is a delay of 48 hours assessing the patient severity.
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E-mail	shanky.mr@gmail.com
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INTRODUCTION Acute pancreatitis is an inflammatory disease of the pancreas that is associated with little or no fibrosis of the gland. The causes are many. Presentation of the disease varies from mild abdominal pain to shock, sepsis, multi organ failure or even death. The exact pathogenesis is not yet clearly understood. This may be due to relative inaccessibility to clinical experimental studies. The usual clinical scenario is a chronic alcoholic presenting with severe epigastric pain radiating to back with vomiting. Most of the patients have hyperamylesia. role of imaging with respect to ultrasound is limited. However, computerized tomography is very useful in diagnosing, assessing the...

CONTENTS

INTRODUCTION

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ELIGIBILITY CRITERIA

All patients admitted and diagnosed as having acute pancreatitis were analysed and considered for study. This was a prospective study undertaken in K.A.P.V. Government Medical College Hospital, Trichy from September 2011 to November 2013.

EXCLUSION CRITERIA

- Chronic pancreatitis patients
- Acute exacerbation of chronic pancreatitis.

AIM OF THE STUDY

1. To assess the severity of acute pancreatits in K.A.P.V. Government Medical College, Trichy.
2. To analyse management and outcome of patients with acute pancreatitis in our hospital.

REVIEW OF LITERATURE

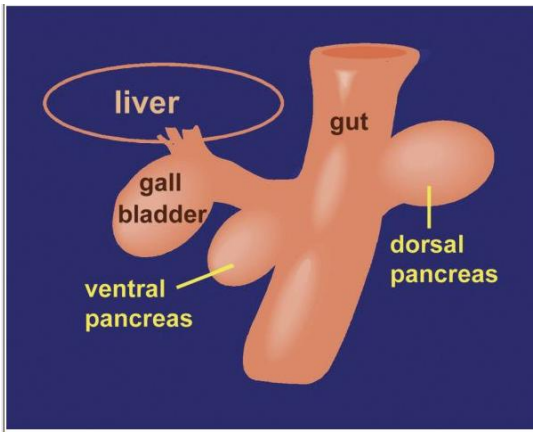
Anatomy, Physiology, and Embryology of the Pancreas[1]

The healthy, mature pancreas functions silently to produce digestive enzymes and hormones. It usually comes to notice only when a pathologic alteration produces symptoms, frequently involving pain.

The pancreas originates during early development of the gastrointestinal system, then differentiates and grows to produce a large gland stretching approximately 15 cm from the duodenum toward the spleen along the posterior body wall. It weighs approximately 90 g normally. The exocrine pancreas produces and releases digestive enzymes into the duodenum. Hormones, including insulin, are released from the endocrine portion into the blood vascular system.

Tracking the development of the pancreas from its first appearance in the primitive gut to the acquisition of its mature form can provide some understanding of its relationship with adjacent structures. (1).

EARLY ANATOMIC FORMATIONS



The pancreas and liver begin as epithelial buds from the primitive gut. Early in embryonic development the endoderm folds into a tube, the anterior part of which is the foregut. At the same time the precursors of major blood vessels form in the surrounding mesenchyme, which is important in early pancreatic development. The primitive aorta, paired anteriorly and fused into a single vessel in the region of the foregut, lies immediately dorsal to the gut. Primitive vitelline veins lie ventral to the gut. The primitive gut and the primitive vessels are, at this stage, tubes consisting solely of a single layer of epithelium. Interaction of the epithelium of the aorta with that of the gut induces proliferation of endodermal epithelium and the beginning of differentiation to form the dorsal primordium of the pancreas . A similar inductive process occurs where vitelline vein endothelium touches gut endoderm to produce ventral buds. The proliferating pancreatic buds express the *Pdx1* gene, providing an early marker of pancreatic differentiation. (1).

Dorsal and ventral pancreatic primordial are formed by the epithelium of dorsal and ventral buds.. The liver, gallbladder also develop from the right ventral primordium. The epithelium expands as primordial tubules that branch as they grow into the surrounding mesenchyme. Mesenchyme is cellular at first. Adult connective tissue is derived from mesenchyme. Cells become more dispersed as differentiation toward adult extracellular matrix proceeds.

Although some differentiation in genetic expression begins, the epithelial cells remain morphologically similar for some time. The branched primordial tubules, which appear as primitive ducts, may reunite within the primordium to form circular continuities. (1)

The pancreas initially grows within a free mesentery, but subsequent changes convert it into a retroperitoneal organ. Rotation of the gut moves the duodenum from the midline to the right side, its middle segment pressed against the dorsal abdominal wall. The pancreas, carried along in the duodenal mesentery, similarly acquires a retroperitoneal position as the mesenteric layers fuse.

Its location at the dorsal abdominal wall just posterior to the diaphragm places the pancreas into close association with a myriad of major vessels and nerves. The aorta gives off the celiac trunk on leaving the thoracic cavity and before passing along the dorsal surface of the pancreas. The superior mesenteric artery runs posterior to the pancreas to pass ventrally over the duodenum. The superior mesenteric vein, paralleling the artery, passes to the dorsal surface of the pancreas where it is joined by the splenic vein to form the hepatic portal vein. The thinner part of the pancreas over the superior mesenteric– hepatic portal vein is its neck, separating the head from the body. (1)

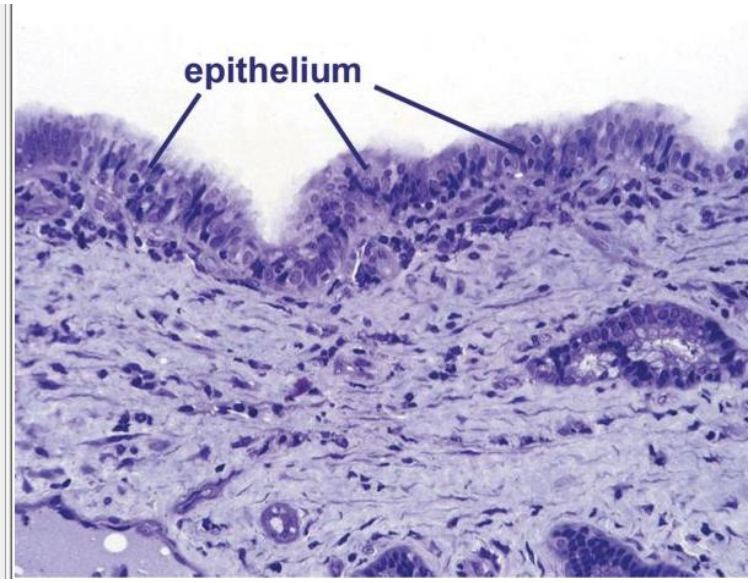
The endocrine and exocrine components of the pancreas derive from the same population of cells. Differentiation of the cells comprising the primitive ducts leads along three main pathways. At intervals cells bud off the primitive ducts, proliferate into spheroidal groups, lose their contact with the lumens, and become the islets of Langerhans. At the ends and along the sides of the primitive ducts, differentiating cells produce the spheroidal and elongate collections that constitute the acini. The remaining cells stay approximately in their original relationship to become the mature ducts.

EARLY CELLULAR CHANGES

Early in embryonic development proliferation and differentiation of primitive duct cells produce the precursors of islets of Langerhans. The islet cells produced include those capable of expressing insulin, glucagon, somatostatin, or pancreatic polypeptide. Cells expressing each of these products are present by 14 weeks' gestational age. During the early stages of islet formation, some of the cells express markers for duct cells at the same time they express insulin; that is, they represent an intermediate stage between duct cells and mature islet cells. The duct markers disappear with increased age.

ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS

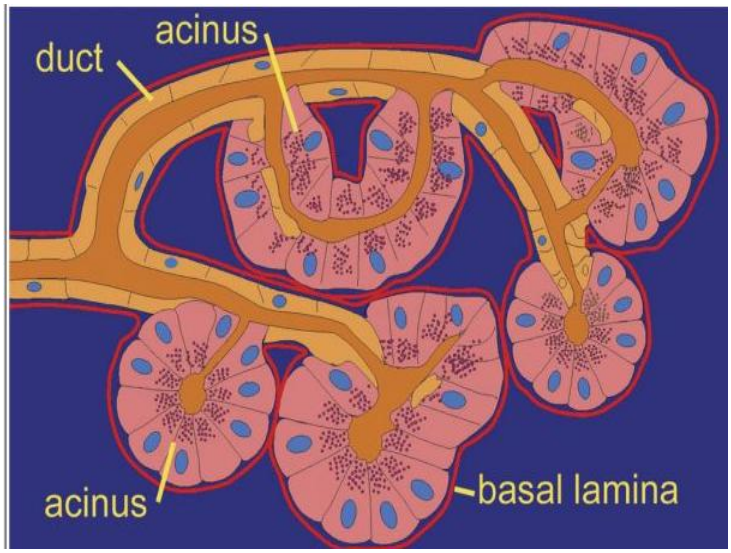
Ductal Epithelial Cells



Mature ducts form a barrier between the secretion products they carry in their lumens and the surrounding extracellular matrix. The epithelium constitutes a continuous layer lining the ducts. Ductal epithelial cells lie side-by-side with the apposing membranes at the luminal surface joined by tight junctions. Therefore interchange of fluid and ions between duct lumen and extracellular space must be a transcellular event regulated by the cells traversed. Larger ducts present a smooth, undulating surface toward the lumen. Tightly joined ductal cells display microvilli projecting into the lumen between tight junctions. In the absence of pancreatic disease, the ducts are surrounded by a connective tissue matrix with a small number of cells. With disease, however, such as chronic pancreatitis, the epithelial barrier may be missing in places, with accumulation of inflammatory cells and

proliferation of blood vessels exposed directly to the lumen. In this condition interchange of substances and cells between lumen and the extracellular space of the pancreas can occur without the regulation normally provided by the ductal epithelium. (1)

Acinar Cells



Acinar tissue constitutes the greatest proportion of the mature normal pancreas. Like islet cells, early acinar cells are derived from primitive ducts. Acinar cells are present by the 4th month of gestation. Enzymes are synthesized in an abundant rough endoplasmic reticulum prominent in the base of acinar cells and packaged in the Golgi apparatus for storage. Many of the enzymes are stored in granules as precursor products (zymogens) mainly in the apex of acinar cells. Stimulation of the acinar cell releases zymogen granules at the cell membrane bordering on the acinar lumen. Activation of the enzymes occurs on entry into the duodenal lumen where enterokinase acts on the pancreatic juice

conducted there through the pancreatic ductal system. Trypsin is activated, in turn activating other zymogens.

Pancreatic ducts branch and rebranch, producing progressively more and smaller ducts. The smallest ducts are intimately associated with acini. Some duct cells form tight junctions with adjacent acinar cells. The result is a continuous lumen, surrounded by a continuous epithelium, from the ductal system through the acinar system. (1)

Dysfunctional Cellular Changes

Imposition of abnormal conditions on the pancreas leads to transdifferentiation, in which fully differentiated cells revert to another cell type. Relative proportions of components change when abnormal situations alter factors modulating cellular behavior. Some of the change can occur by necrosis and apoptosis. If, for example, acinar cells die while other cell types survive, their proportion decreases. A more complex change that occurs is the transition of cells backward along the path differentiation has taken. Acinar and islet cells transdifferentiate to ductal cells. The result is clusters of small ducts, called *tubular complexes*. Tubular complexes may be produced by obstruction of the ductal system or by genetic abnormalities. They are common in chronic pancreatitis and pancreatic cancer. Cell markers and morphology change as acinar cells revert to a ductal phenotype, signaling the expres-sion of different sets of genes. Furthermore, the cells that have secondarily

acquired the ductal phenotype exist in the population that is susceptible to malignant transformation. (1)

Cellular Membrane Function

Pancreatic cells react to many hormones and neurotransmitters. Among the variety of receptors contained in their membranes are receptors for cholecystokinin, secretin, and acetylcholine.

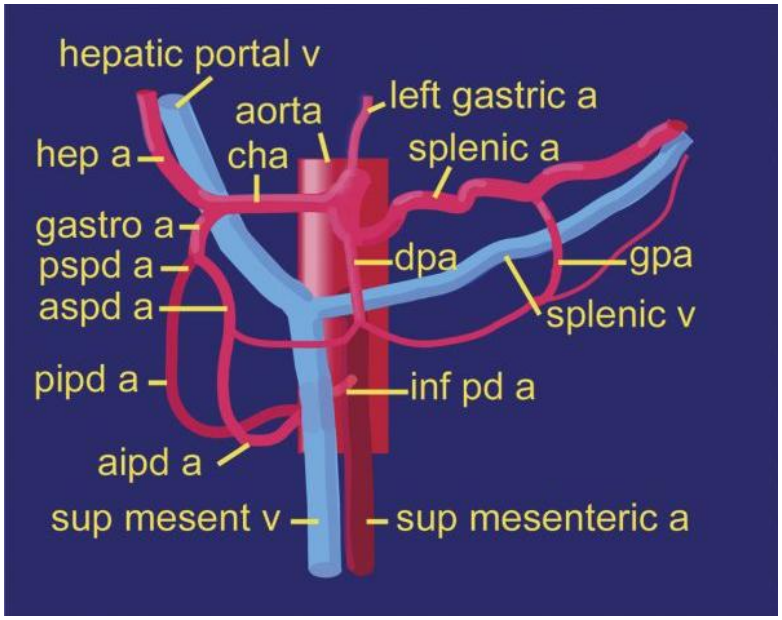
Release of secretin from the duodenum results in the secretion of bicarbonate-rich fluid into the ductal system. Secretin interacts with its receptor on the surface of ductal cells. Bicarbonate, generated within the cell through the action of carbonic anhydrase, is expelled into the duct lumen through the apical membrane via an exchange with chloride. The channel that supplies the lumen with chloride ions is the cystic fibrosis transmembrane conductance regulator that is defective in cystic fibrosis.

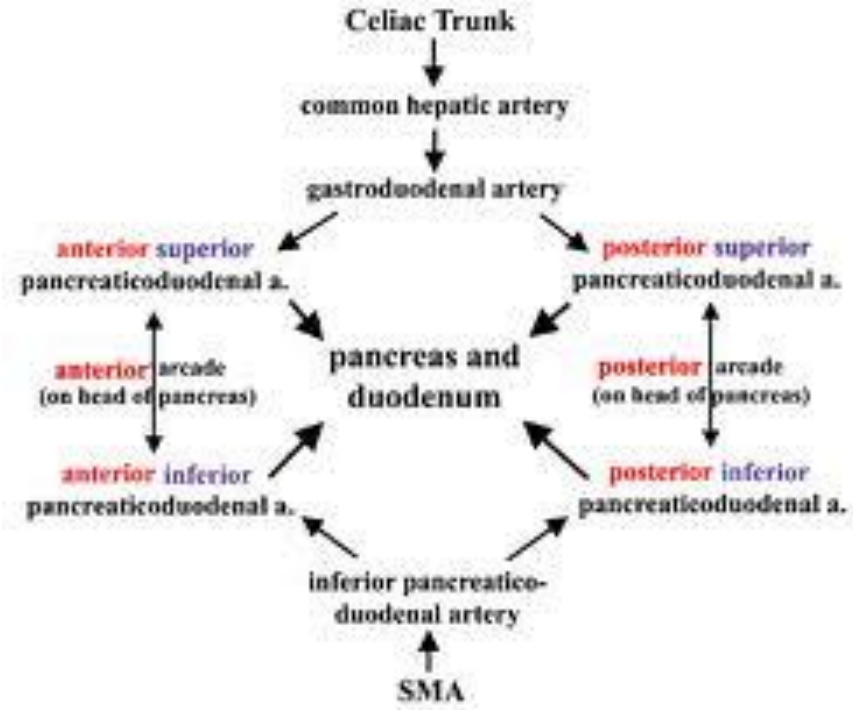
Release of cholecystokinin results in secretion of digestive enzymes from acinar cells. Stimulation of secretion is accompanied by increased concentrations of intracellular calcium ions within acinar cells.The contents of the granules are released by the fusion of membrane of the zymogen granules with the apical membrane. It is likely that the main interaction on the basolateral membrane of human acinar cells is not between cholecystokinin and its receptor but between acetylcholine and its receptor. Human acinar cells do not react to cholecystokinin the same as in some species. Rather, it is likely that receptors for cholecystokinin

on afferent fibers of the vagus nerve are activated, initiating stimulation of pancreatic secretion by vagal cholinergic pathways.

Ligands and receptors normally interact within certain limits of concentration. In unusual situations the limits are exceeded. Supramaximal stimulation of acinar cells with the cholecystokinin, a procedure that produces experimental acute pancreatitis, makes their cell membranes permeable to large molecules, suggesting a mechanism for the initiation of acute pancreatitis. Perhaps overstimulation via the vagus nerve damages acinar membranes, allowing early events characteristic of pancreatitis. (1)

Blood Supply





The abundant blood supply of the pancreas comes from branches of the aorta that also serve adjacent abdominal organs. Arteries originating in the aorta branch to serve liver, stomach, spleen, and intestine in common with the pancreas. The celiac and superior mesenteric arteries constitute the primary arteries from which others are derived. The splenic and common hepatic arteries branch from the celiac. Dorsal and greater pancreatic arteries, in addition to smaller ones, branch from the splenic artery. The gastroduodenal artery gives rise to pancreaticoduodenal artery which anastomoses with the pancreaticoduodenal branches from the superior mesenteric artery.

Perfusion of the islets of Langerhans is much greater than that of acinar tissue. One or more arterioles enter the islet to branch into a

prominent capillary plexus. The fenestrated capillaries of the islets empty directly into the acinar capillary plexus.

Veins of the pancreas drain eventually into the hepatic portal system, so may become involved in the spread of pancreatic cancer to the liver. Cancer may invade the hepatic portal vein along its path dorsal to the pancreas. (1)

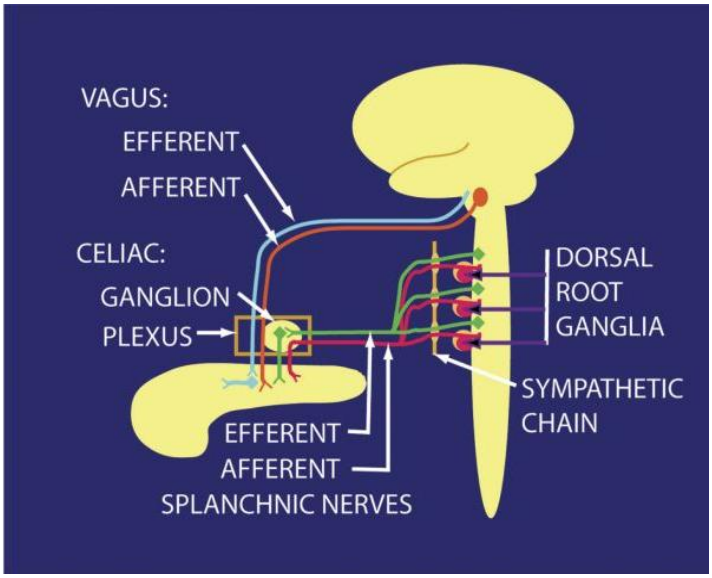
Lymphatic System

Lymphatic vessels lie mostly in the connective tissue septa of the pancreas. They are not particularly numerous, they have thin walls, and they tend to collapse when not in situ, so they are difficult to observe. A few intralobular lymphatic vessels drain into the interlobular plexus. These, in turn, coalesce into larger vessels which tend to parallel the blood vessels serving the pancreas. Lymphatic vessels emerge on the surface of the pancreas to enter lymph nodes. Efferent vessels from multiple nodes empty eventually into the thoracic duct.

In the normal situation the lymphatics carry mostly excess interstitial fluid so could be considered to serve as an overflow. In pathologic situations, other things gain access and are conducted. Lymph-borne metastases of pancreatic cancer are found in primary and secondary lymph nodes interposed between the pancreas and the thoracic duct. Lymph nodes surround the pancreas and lie before and along the sides of the aorta and its branches. Many of the nodes are associated with blood

vessels and may be described according to the vessel. Celiac, splenic, hepatic, gastroduodenal, pancreaticoduodenal, and superior mesenteric groups of nodes are described. Suprapancreatic and infrapancreatic groups lie immediately outside the pancreas. (1)

Nervous System



Sympathetic fibers carried primarily in the splanchnic nerves originate in the intermediolateral cell column of the spinal cord. Accompanying sensory fibers have their cell bodies in the dorsal root ganglia. Parasympathetic fibers are carried with accompanying sensory fibers in the vagus nerve, which is attached to the brain. Sympathetic innervation affects pancreatic vasculature. Parasympathetic innervation modulates secretion. However, normal control of pancreatic function is complex and relies on simultaneous regulated activity of all mechanisms, so that a defect in one component can affect another. Physiologic control of and response by the pancreas is mediated in part by peptidergic

innervation.Among these neurotransmitters are substance P, neuropeptide Y, calcitonin gene-related peptide, and vasoactive intestinal polypeptide.

Splanchnic and vagus nerves pass through a plexus of nerve fibers and ganglia distributed around the base of the celiac artery. The sympathetic fibers synapse on secondary neurons in the celiac ganglia in addition to contributing to the celiac plexus. The parasympathetic fibers and sensory fibers pass through the celiac plexus without synapse. Parasympathetic fibers synapse on cell bodies of secondary neurons that form ganglia within the pancreas. (1)

Nerve fibers combine in the celiac plexus and are distributed to the substance of the pancreas as networks surrounding the arteries of supply. The nerves that are distributed thus are mixed; that is, a nerve may contain sympathetic, parasympathetic, and sensory fibers. The nerves are mainly unmyelinated.

On direct intense stimulation of nerves, or because of damage to the perineurium and nerve fibers by invasion of pancreatic cancer or chronic inflammation accompanying chronic pancreatitis, pain may be induced and sustained. A logical approach to the treatment of unremitting pain is to interrupt the pathway conducting it. Common approaches have been to block the celiac plexus or to interrupt the splanchnic nerves since they are the principal pathways for pain conduction from the pancreas.

The severity and breadth of pain generation may lead to incorporation, at least potentially, of other nerves that are less central to pain conduction from the pancreas. It is possible that sensory nerves in the vagus may contribute to pain generation under appropriate circumstances. The pancreas lies on the posterior abdominal wall, and pancreatic cancer may extend from the pancreas proper to involve spinal nerves. Branches of the phrenic nerve could become involved in pain transmission. (1)

ETIOLOGY[2]

Nearly 90% of all episodes of acute pancreatitis are attributable to gallstone disease and alcohol abuse. Further etiologies of acute pancreatitis and clinical associations are listed in Box. The most common are briefly discussed, including biliary tract stones, alcohol, postprocedural, trauma, hyperlipidemia, hyperparathyroidism/ hypercalcemia, hereditary, pancreas divisum, infection, medications, pregnancy, and idiopathic. (2)

Etiology of Acute Pancreatitis and Clinical Associations

- Biliary tract stone disease
- Ethanol/alcohol abuse
- Trauma
  - Postprocedural
    - Post-ERCP
    - Postoperative
  - Direct blunt trauma
- Hyperparathyroidism/hypercalcemia
- Hyperlipidemia
- Hereditary pancreatitis
- Infections
  - Viral
  - Parasitic
  - Fungal
  - Bacterial
- Mechanical obstruction
  - Tumors
  - Pancreatic divisum
  - Duodenal obstruction
- Medications
  - Antibiotics: sulfonamides, tetracyclines
  - Calcium
  - Cardiovascular: clonidine, quinidine, warfarin
  - Diuretics: furosemide, thiazides, ethacrynic acid, diazoxide
  - Steroids: estrogen, glucocorticoids
  - Other: azathioprine, cimetidine, methyldopa, phenformin
- Pregnancy
- Scorpion *(Tityus trinitatis)* venom
- Idiopathic

Biliary Tract Disease

Although acute pancreatitis is documented in association with acalculous biliary tract disease, bile duct stones (choledocholithiasis) represent the most common form of associated biliary abnormality. The mechanism by which a gallstone may cause pancreatitis is not entirely clear, although gallstones have been implicated ever since Opie made the seminal observation in 1901.This led him to propose the "common-channel hypothesis," in which a blockage below the junction of the biliary and pancreatic ducts would cause bile to flow into the pancreas, which could then be damaged by the detergent action of bile salts.

Important objections to this theory include the anatomic reality that the majority of individuals have such a short common channel that a stone located there would block both the pancreatic and biliary ducts, effectively isolating the two systems. Also, the pressure in the bile duct is lower than the pancreatic duct and hence flow from bile duct to pancreatic duct is less likely.

Another proposed mechanism of causation postulates that passage of a gallstone through the sphincter of Oddi renders it momentarily incompetent, permitting the reflux of duodenal juice containing activated digestive enzymes into the pancreatic ductal system. However, it is questionable whether the transit time through the sphincter of Oddi is long enough to cause sufficient incompetence. Finally, the observation

remains that procedures designed to render the sphincter incompetent, such as sphincterotomy, do not routinely cause pancreatitis.

Therefore, although it is reasonable to dismiss an incompetent sphincter of Oddi as an etiologic factor in acute pancreatitis, it is not as simple to dismiss the role of gallstones. A clinical study showed that 88% of patients with acute pancreatitis passed gallstones in their feces within 10 days of the attack. This is in contrast to only 11% of gallstone patients who did not have pancreatitis, suggesting that the process of passing a gallstone may cause pancreatitis.

This information justifies searching for a more likely causative factor than abnormal bile or duodenal juice backflow into the pancreas. A common phenomenon by which common bile duct stone, tumor or helminthic infection cause pancreatitis is by duct obstruction. Obstruction of the duct causes duct hypertension which leads to leakage of pancreatic juice from the duct into the parenchyma.the low pH in the parenchyma activates the proteases and trypsin. also the colocolization of inactive zymogens and lysosomal hydrolases in ductal hypertension lead to activation of pancreatic enzymes. (2)

Alcohol

Ethanol can induce pancreatitis by several methods. Various proposed theories include spasm of spinchter of oddi, increased secretion of pancreatic juice followed by precipitation, induction of transient

hyperlipedemic state, increased ductal permeability, reduced blood flow to the pancreas and direct toxic effect on pancreas.

Tumors

A tumor can cause pancreatits by blocking ducts leading to ductal hypertension ans extravasation of pancreatic enzymes into the parenchyma.

Iatrogenic Pancreatitis

Acute pancreatitis can be associated with a number of surgical procedures, most commonly those performed on or close to the pancreas, such as pancreatic biopsy, biliary duct exploration, distal gastrectomy, and splenectomy. Acute pancreatitis is associated postoperatively with Billroth II gastrectomy and jejunostomy, in which increased intraduodenal pressure can cause backflow of activated enzymes into the pancreas. However, pancreatitis also can occur in association with surgery that uses low systemic perfusion, such as cardiopulmonary bypass and cardiac transplantation. Acute pancreatitis has been reported to be associated with severe hypothermia, and the hypothermia associated with cardiopulmonary bypass may be similarly causative. It also is possible that atheromatous emboli or ischemia may cause pancreatic injury. Most commonly, endoscopic retrograde cholangiopancreatography (ERCP) results in pancreatitis in 2 to 10% of patients, due to direct injury and/or intraductal hypertension. (2)

Drugs

For practical reasons, it often is difficult to implicate a drug as the cause of pancreatitis. Many drugs can produce hyperamylasemia and/or abdominal pain, and a drug is considered suspect if the pancreatitis-like illness resolves with its discontinuation. Certain drugs are known to be capable of causing acute pancreatitis. These include the thiazide diuretics, furosemide, estrogens, azathioprine, L-asparaginase, 6-mercaptopurine, methyldopa, the sulfonamides, tetracycline, pentamidine, procainamide, nitrofurantoin, dideoxyinosine, valproic acid, and acetylcholinesterase inhibitors.

Infections

Though mumps, coxsackievirus, and *Mycoplasma pneumoniae* are believed to be capable of inducing acute pancreatitis by infecting the acinar cells, none of these agents has been isolated from a diseased pancreas.

Hyperlipidemia

It has been suggested that lipase can liberate large amounts of toxic fatty acids into the pancreatic microcirculation. This could lead to endothelial injury, sludging of blood cells, and consequent ischemic states

Miscellaneous Causes

Hypercalcemic states arising from hyperparathyroidism can result in both acute and chronic pancreatitis; the mechanism most likely involves hypersecretion and the formation of calcified stones intraductally. Also implicated are infestations by *Ascaris lumbricoides* and the liver fluke *Clonorchis sinensis*, which is endemic to China, Japan, and Southeast Asia. These cause Oriental cholangitis, which is associated with cholangiocarcinoma obstructing the pancreatic duct. A dominant gene mutation following Mendelian inheritance is known to result in hereditary pancreatitis. Whitcomb and associates described several families from various parts of the world who have mutations in their cationic trypsinogen gene *PRSS1*, which results in pancreatitis.[32] Additionally, 20 to 45% of patients with pancreas divisum (unfused ducts of Wirsung and Santorini) develop pancreatitis, but the failure of procedures to improve drainage of the lesser papilla in reducing attacks of pancreatitis, as well as the observed lack of ductal dilatation in such patients, argues against pancreas divisum as an etiologic factor, rendering the role of this condition as yet unclear. Other implicated factors include azotemia, vasculitis, and the sting of the Trinidadian scorpion *Tityus trinitatis*. This scorpion's venom has been shown to cause neurotransmitter discharge from cholinergic nerve terminals, leading to massive production of pancreatic juice. Poisoning with

antiacetylcholinesterase insecticides has a similar effect. Finally, no apparent cause can be ascribed to some episodes of acute pancreatitis, and these constitute the group referred to as *idiopathic pancreatitis*. Some of these patients are eventually found to have gallstone-related pancreatitis, which calls for caution in labeling any episode "idiopathic." (2)

CLINICAL PRESENTATION

The spectrum of clinical presentations of acute pancreatitis is broad. Fortunately, 90% to 95% of patients simply experience a mild, self-limiting bout of acute pancreatitis. Though much less common, 5% to 10% of patients develop a severe, life-threatening episode associated with a prolonged hospitalization, intensive care unit (ICU) stay, and increased morbidity.

The cardinal symptom of acute pancreatitis is the insidious onset of constant epigastric pain, which often radiates to the back; however, other symptoms including anorexia, nausea, emesis, abdominal mass, or fever are often present. Patients with severe, necrotizing acute pancreatitis may develop jaundice, hypotension, shock, and signs of retroperitoneal hemorrhage, specifically an ecchymotic discoloration of the flank (Grey Turner's sign), the umbilicus (Cullen's sign), or the inguinal ligament (Fox's sign). (2)

DIAGNOSIS

Laboratory Tests

Routine blood investigations namely complete blood count, renal function tests, liver function tests, blood sugar, serum amylase are usually done. Hemoconcentration greater than 44%, elevated renal parameters, CRP >150mg/ml often indicate poor prognosis. (8)

Serum amylase and lipase remain the most widely used laboratory tests. The sensitivity of amylase is relatively low since there are multiple causes of hyperamylasemia unrelated to acute pancreatitis. However, concurrently increased lipase and amylase in the setting of abdominal pain increases the sensitivity and specificity of diagnosing acute pancreatitis to 90% and 95%, respectively. Although diagnostic, serum amylase and lipase levels have not been found to correlate with pancreatitis severity. Fractionation of plasma amylase isoenzymes and the calculation of fractional amylase excretion may be helpful in patients with nonpancreatic sources of hyperamylasemia or with renal failure. Nevertheless, the advent of computed tomographic (CT) scan has rendered these latter tests nearly obsolete. (2)

Hyperamylasemia Unrelated to Acute Pancreatitis

- Abdominal
  - Acute appendicitis
  - Biliary tract disease and gallstones
  - Intestinal obstruction and ischemia
  - Liver diseases
  - Pancreatic fistula
  - Peritonitis
  - Pregnancy
  - Perforated viscus
- Impaired amylase secretion
  - Renal dysfunction
  - Nephrolithiasis
  - Macroamylasemia
  - Bisalbuminemia
- Metabolic disorders
  - Diabetic ketoacidosis
- Salivary gland disorders/injury
  - Calculi
  - Hypersecretion
  - Irradiation sialadenitis
  - Mumps
  - Parotitis
- Trauma
  - Burns
  - Cerebral trauma
  - Multiple trauma

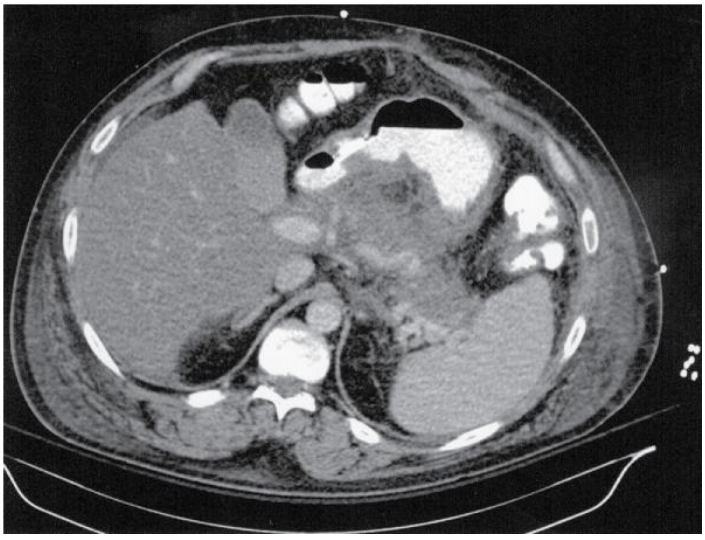
Radiologic Studies

Chest x ray and abdominal x ray erect view may show dilated ileal loops, abrupt cut off of transverse colon, pleural effusion, lung collapse or rarely calcification in the pancreas.

Ultrasonographic Evaluation

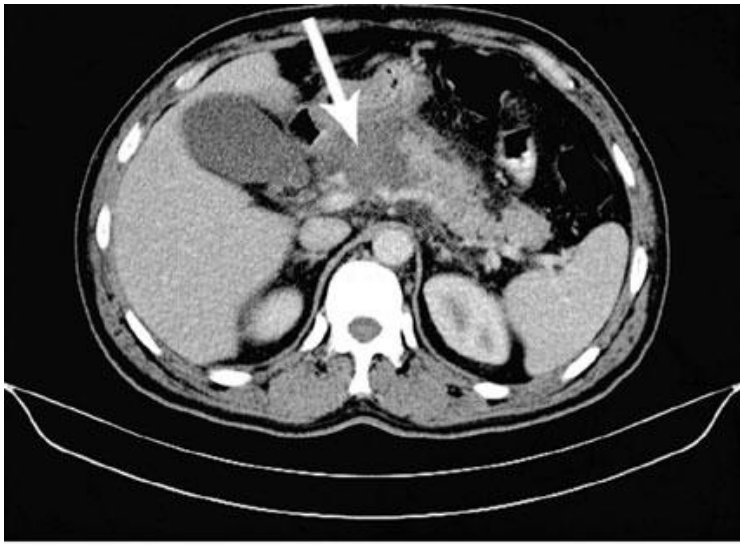
Ultrasound abdomen may show diffused enlarged gland with acute fluid collecton but role of ultrasound is limited because of the overlying gas shadow. Also the retroperitoneal fluid collection may not be properly visualised by ultrasound.

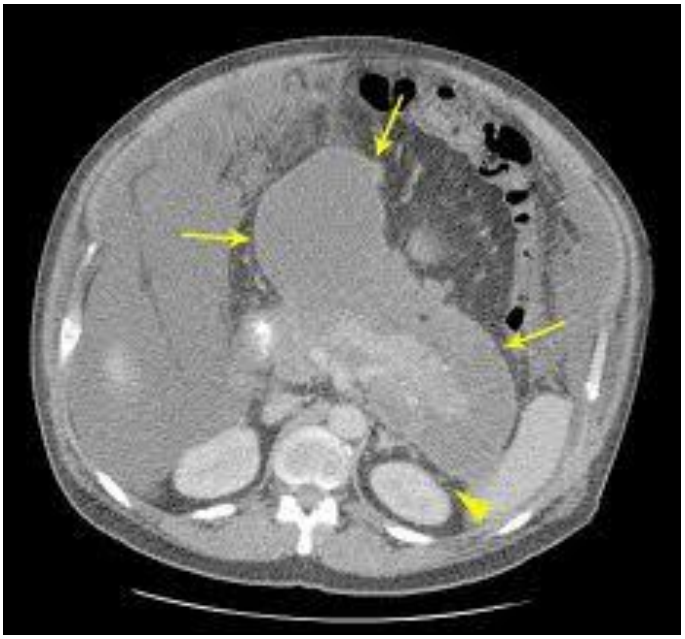
CT Abdomen.

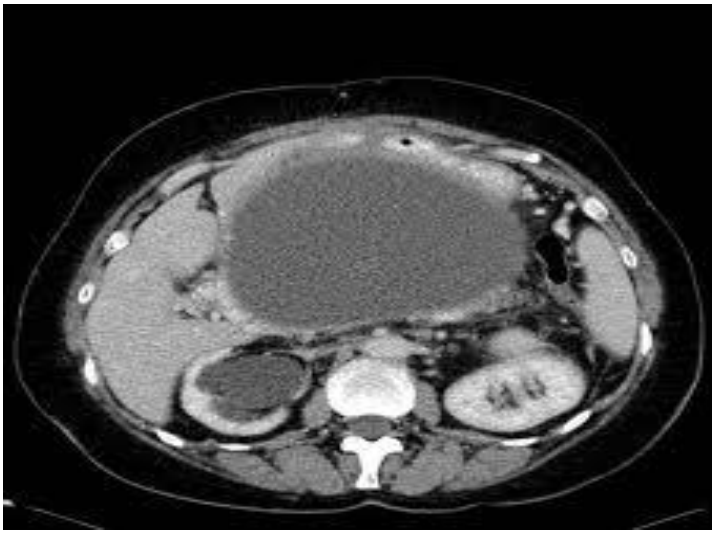


Computerized tomography of abdomen is considered the GOLD STANDARD in diagnosis of acute pancreatitis. In patients with normal renal parameters, around 100 – 150 ml of intravenous contrast (8 – 10 mm collimation) at a rate of 3ml/second is injected. The rationale behind this is in patients with normal pancreas and mild interstitial pancreatits,

the capillary network is intact and the contrast uptake also is uniform resulting in uniformly enhanced gland. In patients with severe pancreatitis where there is destruction of the gland, the uptake is patchy. Diffuse or localized areas of non enhanced areas are considered as pancreatic necrosis. Based on gland necrosis a scoring system is made and the prognosis of the patient is known. The sensitivity of CT is 87% and specificity is 100%. (9). The correlation of CT grading and mortality is patients who have a severity index of 0 or 1 exhibited a 0% mortality rate and no morbidity, while patients with severity index of 2 have no mortality and a 4% morbidity rate. In contrast, a severity index of 7–10 yielded a 17% mortality rate and a 92% complication rate. CT is taken usually on third day from clinical onset of pancreatitis attack because zones of tissue liquefaction become better defined and more easily recognized 2–3 days after the initial onset. Certain pitfalls of CT scan should also be borne in mind. CT taken within 24 -48 hours of onset of pancreatitis may not be useful. Interstitial fliud collection, interstitial edema, fatty infiltration into pancreas may sometimes be misinterpreted as pancreatic necrosis. Retro pancreatic fat necrosis cannot be diagnosed or quantified using CT scan. (3)







CT Severity Index

The CT severity index is an attempt to improve the early prognostic value of CT in cases of acute pancreatitis . Patients with grade A–E pancreatitis are assigned zero to four points plus two points for necrosis of up to 30%, four points for necrosis of 30%–50%, and six points for necrosis of more than 50%. (3)

CT SEVERITY INDEX AND MODIFIED CT SEVERITY INDEX

Characteristics	**CTSI (0–10)**	**MCTSI (0–10)**
Pancreatic inflammation		
Normal pancreas	0	0
Focal or diffuse enlargement of pancreas	1	2
Peripancreatic inflammation	2	2
Single acute fluid collection	3	4
Two or more acute fluid collections	4	4
Pancreatic parenchymal necrosis		
None	0	0
Less than 30%	2	2
Between 30% and 50%	4	4
More than 50%	6	4

Modified CT severity index has Extrapancreatic complications which include One or more of pleural effusion, ascites, vascular complications, parenchymal complications, or gastrointestinal tract involvement. This is given 2 points. (8)

Magnetic Resonance Imaging

In patients with elevated renal parameters and allergic reactions to contrast, MR can be used. Gadolinium-enhanced T1-weighted gradient-echo MR images can depict pancreatic necrosis as areas of nonenhanced parenchyma. T2- weighted images can accurately depict fluid collections, pseudocysts, and areas of haemorrhage. (8)

PROGNOSTIC INDICATORS

Ranson's criteria are the most commonly used scoring system. Patients with one or two criteria have a predicted mortality of less than 1% compared to patients with three criteria (10%) or four criteria (15%); with more than seven criteria, the predicted mortality approaches 50%. Disadvantage of this criterion is that we have to wait for 48 hours to completely assess the patient.

Ranson's Criteria	Nonbiliary Acute Pancreatitis	Biliary Acute Pancreatitis
Admission		
Age (yr)	>55	>70
WBC count (×1000/mm^3)	>16	>18
Glucose (mg/dl)	>200	>220
AST (IU/L)	>250	>250
LDH (IU/L)	>350	>400
Within 48 Hours of Admission		
Hematocrit decrease (points)	>10	>10
BUN increase (mg/dl)	>5	>2
Base deficit (mEq/L)	>4	>5
Fluid replacement (L)	>6	>4
PaO_2 (mm Hg)	<60	<60
Calcium (mg/dl)	<8	<8

AST, aspartate aminotransaminase; BUN, blood urea nitrogen; LDH, lactate dehydrogenase; WBC, white blood cell.

Modified Glasgow criteria are based on eight clinical and laboratory parameters measured within 48 hours of admission. Its limitations are similar to Ranson's criteria.

Modified Glasgow Criteria: Within 48 Hours of Admission

Criteria	Value
Age (yr)	>55
WBC count (×1000/mm^3)	>15
Glucose (mg/dl)	>180
BUN (mg/dl)	>45
LDH (IU/l)	>600
Albumin (g/dl)	<3.3
Pa_{O_2} (mm Hg)	<60
Calcium (mg/dl)	<8

BUN, blood urea nitrogen; LDH, lactate dehydrogenase; WBC, white blood cell.

Acute Physiology and Chronic Health Evaluation (APACHE)-II scoring system incorporates 12 physiologic and laboratory parameters as well as age and comorbid conditions to estimate severity of any disease process. Specifically, a score greater than 9 signifies severe, acute pancreatitis. The APACHE-II scoring system overcomes the shortcomings of Ranson's criteria such that it can be determined on a daily basis. (4)

a: acute physiology score (12	high abnormal rage					low abnormal range			
Physiological Variables	+4	+3	+2	+1	0	+1	+2	+	+4
Temperature – rectal (°C)	≥41	39-40.9		38.5–	36–38.4	34–	32–33.9	30–	≤29.0
Mean arterial pressure (mm hg)	≥160	130–159	110–129		70–109		50–69		≤49
heart rate-ventricular response	≥180	140–179	110–139		70–109		55–69	40–	≤39
Respiratory rate non ventilated	≥50	35–49		25–34	12–24	10–11	6–9		≤5
Oxygen: A – a DO or PaO (mm hg) FiO_2 ≥ 0.5	≥500	350-499	200–349		<200 PO >70	PO 61–70		PO 55–60	PO <55
Arterial ph	≥7.7	7.6–7.69		7.5-7.59	7.33–		7.25–	7.15–	<7.15
Serum hCO – only if no ABGs	≥52	41.5–1.9		32–40.9	23–31.9		18–21.9	15–	<15
Serum sodium (mmol/I)	180	160–179	155–159	50–154	130–149		120–129	111–	≤110
Serum potassium (mmol/I)	≥7	6–6.9		5.5–5.9	3.5–5.4	3–3.4	2.5–2.9		<2.5
Serum creatinine (umol/l)	≥350	200–340	150–190		60–140		<60		
haematocrit (%)	≥60		50–50.9	46–49.9	30–45.9		20–29.9		<20
White Blood cell court (x1000	≥40		20–39.9	15–19.9	30–14.9		1–2.9		<1
Glasgow Coma Score (GCS)	Score = 15 minus actual GCS								

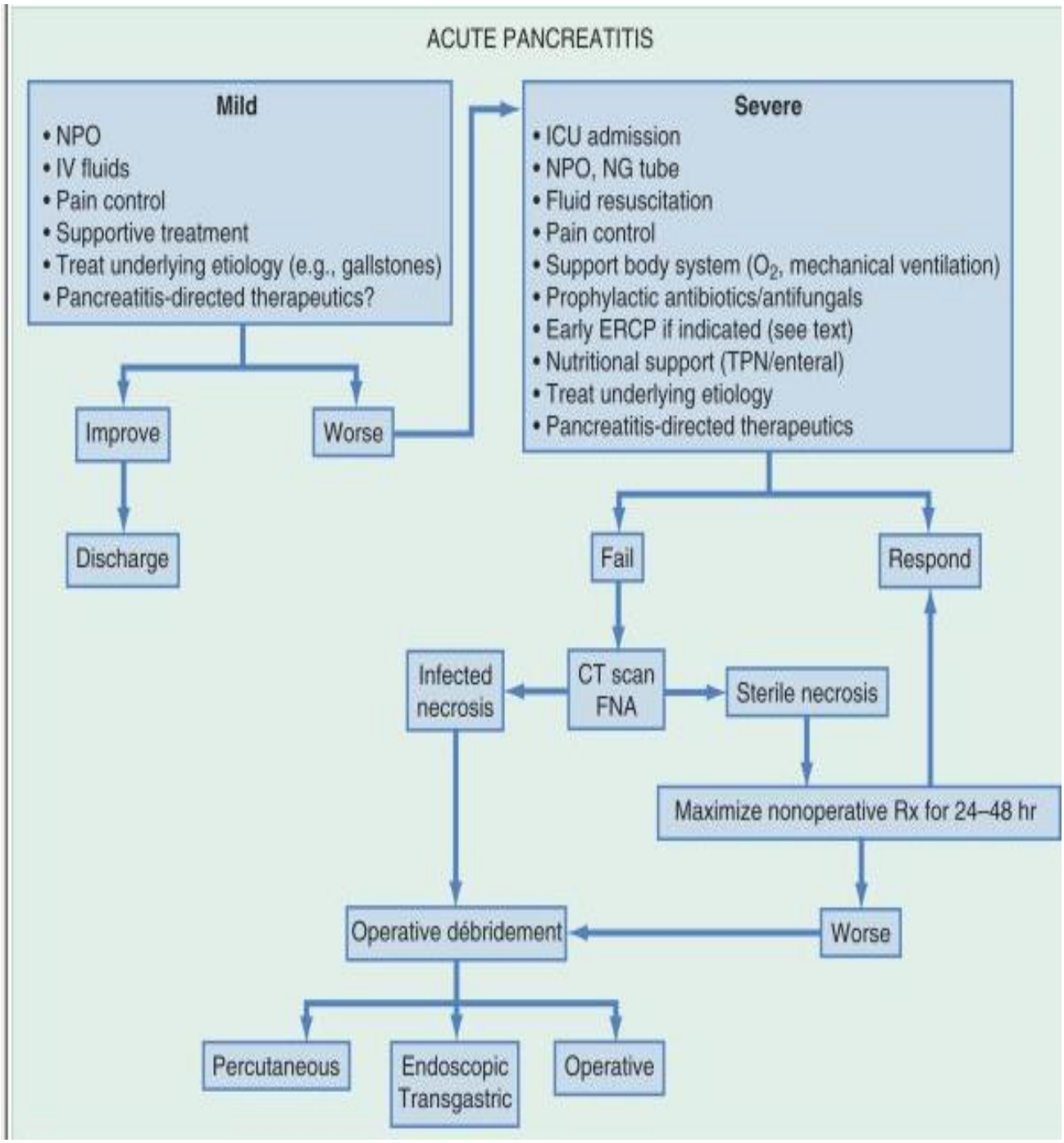
B. age points					
age years	Points	History	Points for elective surgery	Points for emergency surgery	apache ii score:
≥ 44	0	Liver: Biopsy proven cirrhosis and documented portal hypertension or prior	2	5	A: APS score
45–54	2	Cardiovascular NYhA class IV	2	5	B: Age Points score
55–64	3	Respiratory eg. Severe COPD, hypercapnia, home O2 pulmonary	2	5	C: Chronic health points score
65–74	5	Renal chronic dialysis	2	5	
≥ 75	6	Immunocompromised	2	5	Total apache II

APACHE II score of more than 10 are high risk patients. These patients benefit from treatment in an ICU and it is worth while repeating the scores everyday to monitor the clinical state in these patients, in order to detect complications and to institute therapeutic modifications and also to monitor the efficacy of the treatment which is being instituted

The APACHE II[4] system is superior to other systems like Ranson's, because it is the only system which takes into account all the major risk factors that influence the outcome from the disease including the acute physiological derangements, as well as the patient ability to recover which may be diminished by advancing age or chronic disease. Another advantage is that it can be calculated immediately after admission and can be repeated everyday, unlike other scoring systems for acute pancreatitis. The range of the APACHE II score is wide, providing a better spread between the mild and severe attacks because varying weights are assigned to increasingly abnormal values, rather than all or no judgements (4)

Multiorgan Dysfunction Score (MODS)[8] is similar to APACHE-II; this organ-injury based scoring system has been used to predict disease severity. When applied to acute pancreatitis, a score higher than 2 predicts early mortality. (8)

Principles of Management



Resuscitation and Monitoring[5]

Patients with acute pancreatitis should be agressvely resuscitated with intravenous fluids at the rate of 150 - 200ml/hour. Inadequate resuscitation leads to renal insufficiency and increased pancreatic necrosis. However, care must be taken not to over load the patient for the fear of pulmonary edema. Strict input output chart has to be maintained on houly basis and urine output should be maintained more than 0.5ml/kg/hour.

The old teaching was to keep the patient nil per mouth by inserting a ryles tube. In this way adequate rest is given to the pancreas and paralytic ileus is also dealt with. But new teaching is that all patients with pancreatitis need not be kept nil per oral. If there is severe vomiting and patient is in semi conscious state then ryles tube should be inserted to prevent aspiration pneumonia. Patients can be started with sips of water after 2 – 3 days if tolerated and gradually intake can be increased. In patients with severe pancreatitis and those who do not tolerate liquids, total parenteral nutrition may be an option. The disadvantages of TPN is high cost, catheter related complications and alteration in the intestinal mucosal barrier which may lead to further complications. (5)

ERCP

ERCP definitely has a role in the presence of on-going biliary obstruction and cholangitis. There is no role in the absence of biliary obstruction. In fact, ERCP may be harmful in such cases.

Prophylactic Antibiotics

Authors, year	No. of antibiotics/ control	No. of institutions	Antibiotics	Outcomes	Strengths/weaknesses
Isenmann et al., 2004	58/56	19	Ciprofloxacin, metronidazole	No difference in pancreatic infections or mortality	Prospective, randomized, adequately powered
Luiten et al., 1995	52/50	16	Cefotaxime, colistin, amphoterecin B, norfloxacin	Decreased local infections and mortality	Utilized selective digestive decontamination
Sainio et al.,	30/30	1	cefuroxime	No difference in pancreatic infections, sepsis; decreased mortality	Underpowered, single centre
Pederzoli et al.,	33/41	6	imipenem	Decreased local infections and sepsis; no difference in mortality.	Unblended, underpowered, subgroup analysis.

The role of antibiotics is much debated topic. The common organisms involved are E. coli, klebsiella, pseudomonas, staphylococcus and streptococcus. Rationale for using antibiotics is sepsis and MODS develop from local infection and hence by controlling the local infection, sepsis may be controlled. Antibiotics is of use only in early periods where the necrosis is less than 50%. The effectiveness of antibiotic also depends upon the ability of the

drug to penetrate into the pancreas. Also there is a danger of development of antibiotic resistant organisms and fungal infections. The optimal duration of antimicrobial therapy has not been defined, although the incidence of pancreatic infection increases for approximately the first 3 weeks after diagnosis. A treatment course of 1–4 weeks therefore is recommended commonly. Prophylactic use of antifungals is still not yet clear. (5)

Admission to an intensive care unit:

Mortality is generally a consequence of MODS. In the first two weeks, this risk is mainly related to a systemic inflammatory response. Then, mortality is usually associated with pancreatic necrosis and infection. Intensive care unit admission must be considered under the following circumstances:

(1) Persistent MODS for more than 48h and early onset (during the first week) because it is associated with 50% mortality;

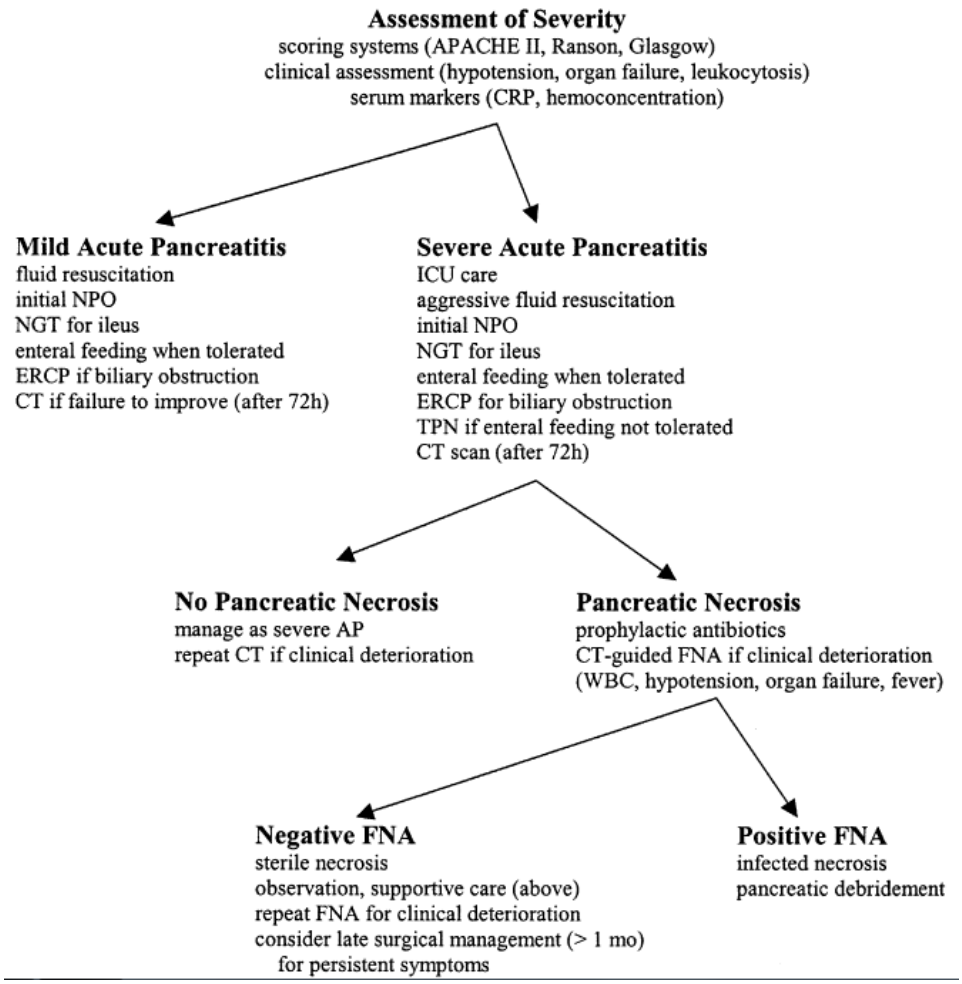
(2) Clinical manifestations predicting MODS development, according to clinical status, multiparametric systems (more than 3 Glasgow or Ranson analytical criteria at 48h or APACHE Ⅱ higher than 8), biochemical data (riboflavin carrier protein >150 mg/dLat 48h), radiological data (persistent pleural effusion for more than 48h after admission) or associated obesity;

(3) Development of local complications.

Surgical Management:

Most of the cases are managed conservatively. The following are the indications for surgery.

1. Infected pancreatic necrosis.
2. Intra-abdominal catastrophe such as perforated viscus .severe sterile necrosis.
3. Symptomatic organised pancreatic necrosis.
4. Diagnostic uncertainity.



Infected Pancreatic Necrosis

The majority of deaths from acute pancreatitis occur in patients with infected pancreatic necrosis. The mortality rate is virtually 100% without intervention, although with appropriate surgical therapy it should approach the less than 15% mortality seen with sterile necrosis. A minority of patients with infected pancreatic necrosis may demonstrate radiographic evidence of such, emphysematous pancreatitis, or intraparenchymal gas. In most patients, CT-guided percutaneous FNA is needed to diagnose infection. As noted previously, both severe sterile necrosis and infected pancreatic necrosis are associated with significant leukocytosis and fever, making clinical distinction impossible. Patients with severe pancreatitis or organ failure or those who fail to improve clinically in the first 2 weeks should be investigated for possible infected necrosis. (5)

Severe Sterile Pancreatic Necrosis

Necrosis of more than 50% may be considered as an indication for surgery but it is delayed till 3 weeks. There is no role of early surgery in acute pancreatitis.

Organized Pancreatic Necrosis

Some patients have persistent pain, malaise or inability to eat. Warshaw described this as persistent unwellness which in pathologic terms is organized pancreatic necrosis. There is clear demarcation from healthy pancreatic tissue and hence surgery in this condition should be delayed upto 3 – 4 weeks.

Pancreatic Necrosis

Pancreatic *necrosis* is defined as a diffuse or focal area of nonviable pancreatic parenchyma that typically is associated with peripancreatic fat necrosis. Necrosis can be sterile or infected. Infected pancreatic necrosis is the leading cause of death associated with severe acute pancreatitis.

The risk of infected necrosis increases with the duration of illness and the extent of necrosis. The risk of infection increases from 24% by the end of the first week of illness, to 36% at the end of the second week, and to 71% by the end of the third week.

There are five routes by which bacteria can infect pancreatic necrosis: (1) hematogenous through mesenteric vessels to the portal circulation, (2) transpapillary reflux of enteric content into the pancreatic duct, (3) translocation of intestinal bacteria and toxins via the mesenteric lymphatics to the thoracic duct and the systemic circulation, (4) reflux of bacteriobilia via a disrupted pancreatic duct into the necrotic parenchyma, and (5) transperitoneal spread. (5)

Timing of Surgery

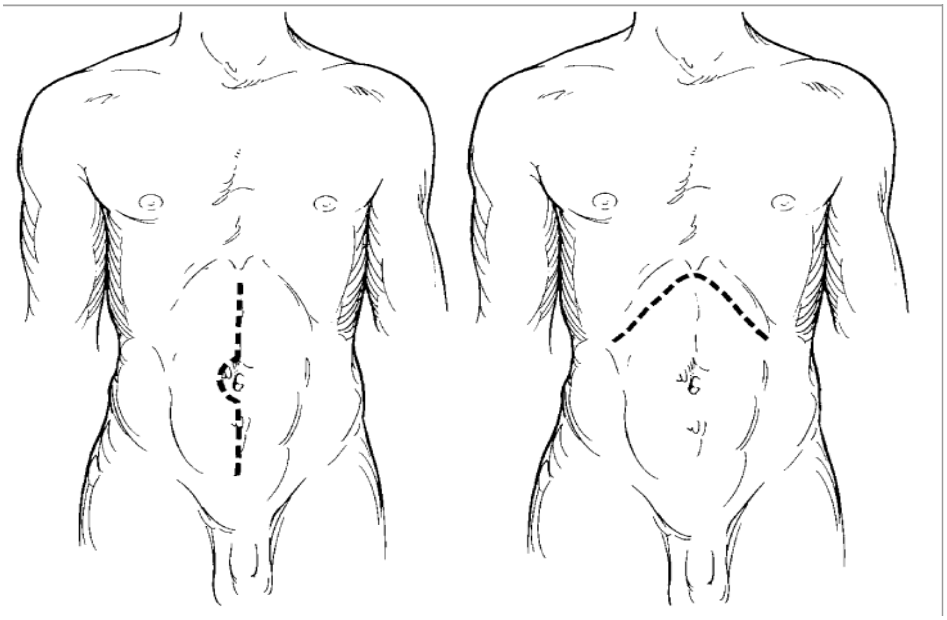
Surgery is usually done 3- 4 weeks after the onset because the demarcation of viable from nonviable tissue is better and chances of bleeding are less.

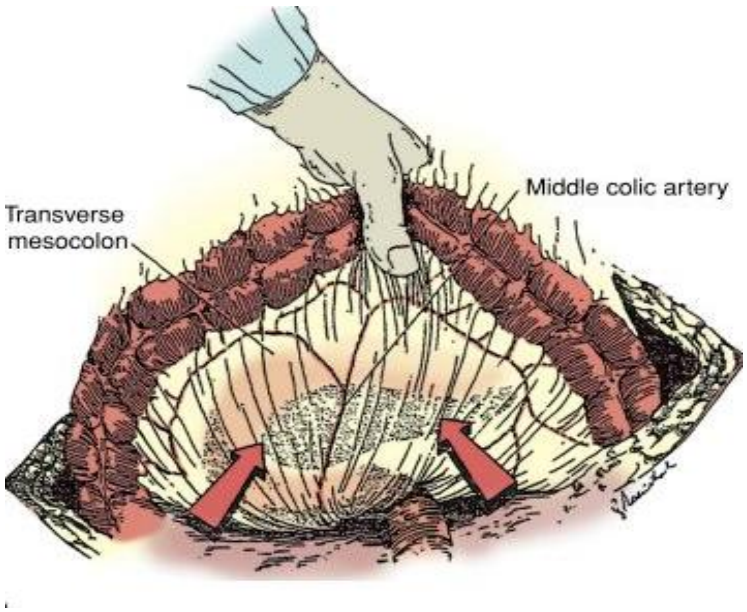
Percutaneous Drainage[6]

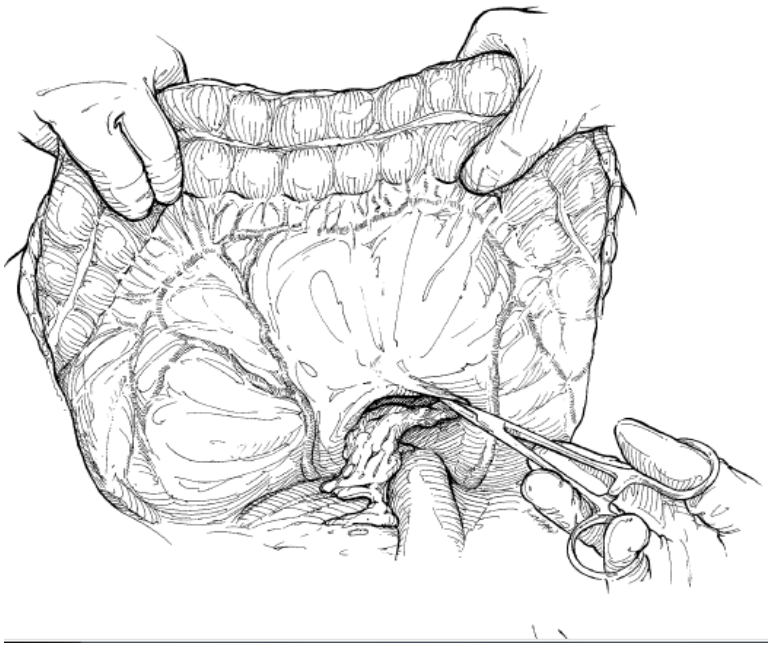
There are two settings in which percutaneous drainage is useful. The first is in an unstable septic patient with evidence of a tense rim-enhanced collection

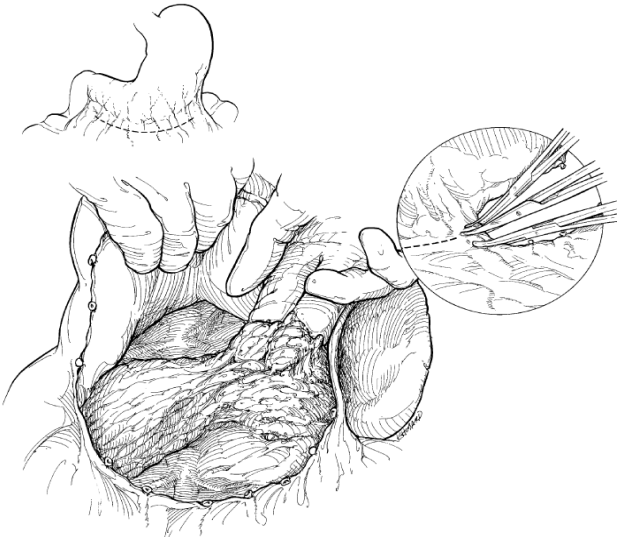
(pancreatic abscess) with a significant fluid component on CT scanning. The second setting in which percutaneous drainage is important is to establish the optimal route for dilatation and subsequent percutaneous necrosectomy, should this be appropriate. This will require careful discussion between the radiologist and surgeon. It usually involves a left-flank puncture and a route along the axis of the body/tail of the pancreas. (6)

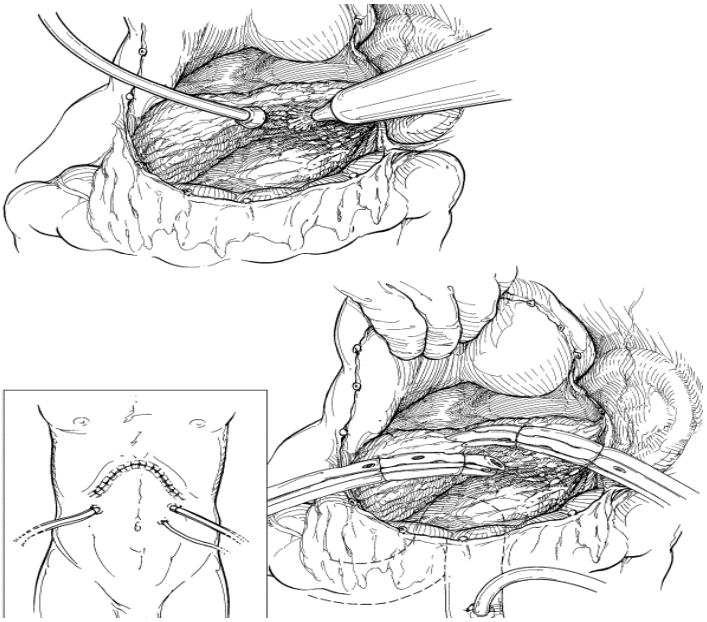
Surgery for Infected Necrosis





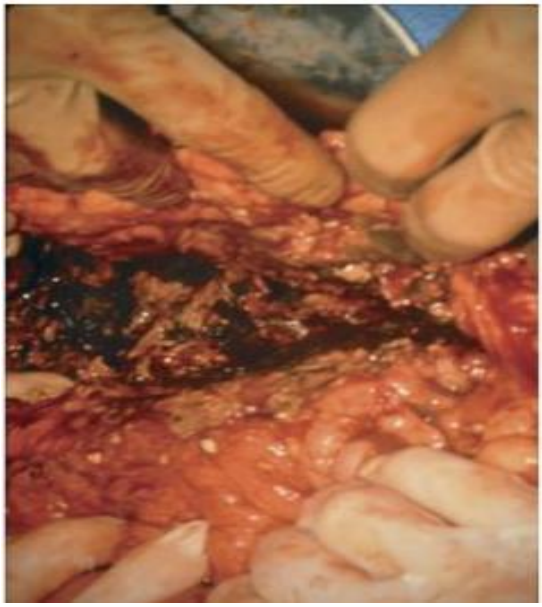






Pancreatic resection is a historical approach that has been associated with unacceptable complication and mortality rates. Pancreatic necrosectomy involves removing the devitalized pancreatic and peripancreatic tissue and drainage of associated pus. The usual approach to the pancreas is through the gastrocolic omentum into the lesser sac. Sometimes it is easiest to enter the region through the transverse mesocolon, from the greater sac, and to the left of the DJ flexure. At the same time, it is useful to take down both colonic flexures, providing better exposure and reducing the risk of subsequent injury to the colon from tube drains. The head of the pancreas then can be approached anteriorly and posteriorly (after kocherization). If the abdomen is opened though a bilateral subcostal incision, in line with the opening to the lesser sac, subsequent laparotomies do not need to disturb the greater peritoneal sac or the

upper abdomen. It is not necessarily a one-stage procedure, especially if an early necrosectomy is embarked on. Necrosectomy is a careful process, best accomplished by an educated finger. The extent of the cavity can be explored and the gentle separation of necrotic material accomplished. Necrotic extensions from the primary cavity need to be explored, often into the root of the small bowel mesentery and down the retrocolic gutters. Care must be taken to remove only what easily separates and to avoid injury to major vessels. The removal of necrotic material may be assisted by irrigation, pulsatile irrigation, gauze, and sponge forceps. When contained by a mature wall, it is advisable to avoid opening up the area too widely. The next step is placement of large-bore, soft, dependent drains to cover all the regions of what is often a complex area. Continuous lavage with peritoneal dialysis fluid, at flow rates of 300–1000 mL/h, may reduce the need to reoperate and often is continued for 2–3 weeks. The most common postoperative complications are hemorrhage and fistulization (pancreas, small and large intestine). The use of packing is lifesaving for major hemorrhage that occurs at the time of necrosectomy, but when used routinely, it is associated with a higher incidence of enteric fistulas. (6)



Prognosis

The prognosis of patients with necrotizing pancreatitis depends on the extent of necrosis and the onset of infection. The overall mortality associated with pancreatic necrosis is 30–40%. This is an average of two major components—the mortality associated with sterile necrosis, which is 0–11%, and that for infected necrosis, which is usually 20–40% but may reach as high as 70%. Most deaths are in the context of multiorgan failure.

Pancreatic Abscess

The Atlanta definition is a circumscribed intra-abdominal collection of purulent material containing little or no pancreatic necrosis. In contrast, infected necrosis is characterized by the extension of necrotic tissue in the pancreas and peripancreatic and/or retroperitoneal regions. The distinction between infected necrosis and pancreatic abscess is of importance because they are managed differently. The predominantly liquid contents of an abscess may be treated by percutaneous drainage, whereas the solid contents of infected necrosis will require debridement. (6)

Diagnosis

The clinical hallmarks of abscess are well recognized, with the development of fever, tachycardia, abdominal pain, and leukocytosis. Although these features may be present already, abscess formation often is accompanied by a secondary deterioration in the patient's clinical course. A secondary elevation in serum CRP level also may occur. Confirmation of a pancreatic abscess is best obtained by a contrast-enhanced CT scan. A rim-enhancing fluid collection arising from a region of previous pancreatic necrosis or pseudocyst is highly suggestive. The presence of bacterial (and fungal) infection can be confirmed by CT-guided FNA.

Treatment

If it is a pure pancreatic abscess with little or no necrosum, then percutaneous drainage may be done. However, the risk of developing pancreatic fistula is high. Hence percutaneous drainage through stomach with subsequent internelixation with a mushroom or J stent may be done. If it is bulging into the stomach or duodenum, endoscopy followed by drainage into stomach is done. (6)

Hemorrhagic complications:

Causes may include gastroduodenitis secondary toadjacent inflammation, rupture of pseudocyst, erosion of splenic artery or associated bleeding peptic ulcer. Diagnosis is by endoscopy and CT Angiography. Definitive therapy includes embolization or surgery.

Pancreatic ascites and pancreatic effusion:

Also termed pancreatic internal fistula, is usually due to chronic leakage from pseudo cyst secondary to alcoholic pancreatitis in adults or traumatic pancreatitis in children. Weight loss and abdominal distension are the features. May be diagnosed by ERCP. Prolonged nil per oral, nasojejunal feeding and stomatostation uaually works. Rarely stenting of the duct may be required. If the distal pancreas is involved, then distal pancreratectomy and internal drainage like pancreatico jejunostomy is done.

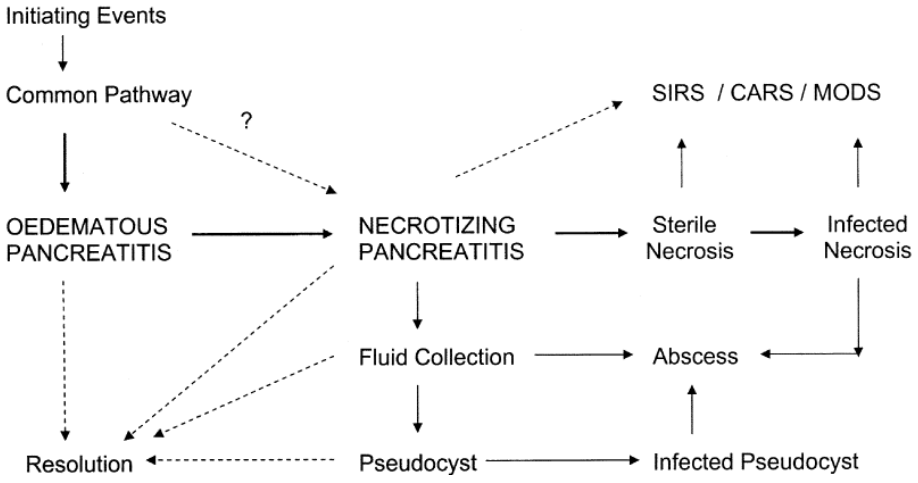
Abdominal and retroperitoneal collections:

Collections in the retroperitoneum depends upon the nature of the collection, time since collection, communication with the main pancreatic duct. For collection less than 4 weeks, a pigtail catheter may be inserted(keeping in mind the risk of introcing an infection). Those communicating with main pancreatic duct may be stented. Those with semisolid debris may have to be evacuated surgically.

Thrombosis of splenic vein and portal vein:

Relatively rare. Subsides spontaneously after inflammation redeces. Main portal vein involement may warrant use of anti platelet drugs but the risk of intrapancreatic bleeding has to be kept in mind.

PANCREATIC PSEUDO CYSTS:

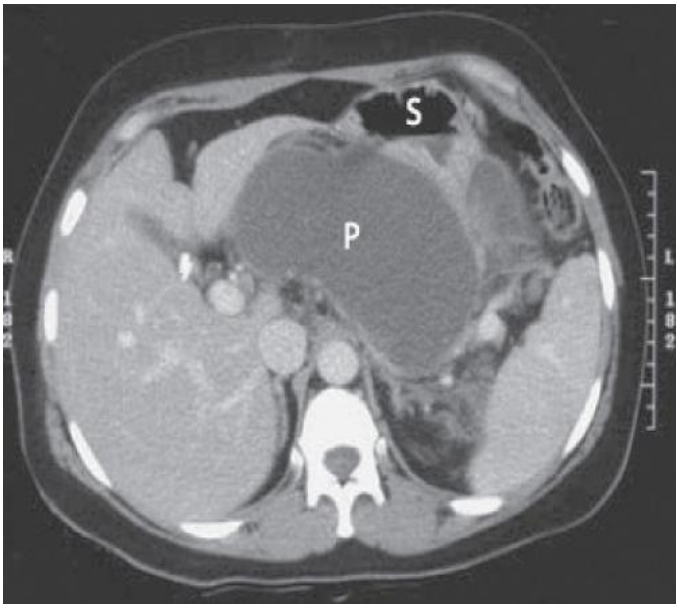


It is defined as peri pancreatic fluid collection contained by granulation tissue which does not have a epithelial lining (in contrast to pancreatic cystic neoplasm which has a epithelial lining).following an episode of acute

pancreatitis, most of the pseudo cysts are located near the head region in the lesser sac but may also be formed in mediastinum, thorax, pelvis or scrotum.

PATHOGENESIS OF PSEUDO CYSTS:

Pseudo cyst formation requires either pancreatic duct disruption or pancreatic duct obstruction. When there is disruption of the duct, the leakage of enzyme rich contents in the peritoneum incites marked inflammation. After a period of 4 – 6 weeks, the collection is contained by granulation tissue. The size of the cyst may remain static or enlarge if it communicates with the main pancreatic duct. Contents is usually sterile clear liquid. Haemorrhage causes xanthochromia. Necrotic material may be present after acute pancreatitis. Pseudo cysts may rupture causing ascites, or may compress onto the surrounding organs or may rupture causing pancreatico pleural/bronchial fistula. Pseudo cysts following trauma are usually located anterior to pancreas as the duct crosses the vertebra anteriorly. In chronic pancreatitis, pseudo cysts are formed due to duct blow outs. There is usually a stricture in the distal duct with the proximal duct communicating with the cyst. (6)



The D'egidio Classification of Pancreatic Pseudocysts and the Primary Treatment Options (6)

	Context	**Pancreatic duct**	**Duct-pseudocyst communication**	**Primary treatment**
Type 1	Acute postnecrotic Pancreatitis	Normal	**No**	Percutaneous drainage
Type 2	Acute-on-chronic Pancreatitis	Abnormal (no Stricture)	**50:50**	Internal drainage or Resection
Type 3	Chronic pancreatitis	Abnormal (stricture)	**Yes**	Internal drainage with duct Decompression

Complications of psedocysts:

1. Compression of stomach, duodenum, common bile duct.

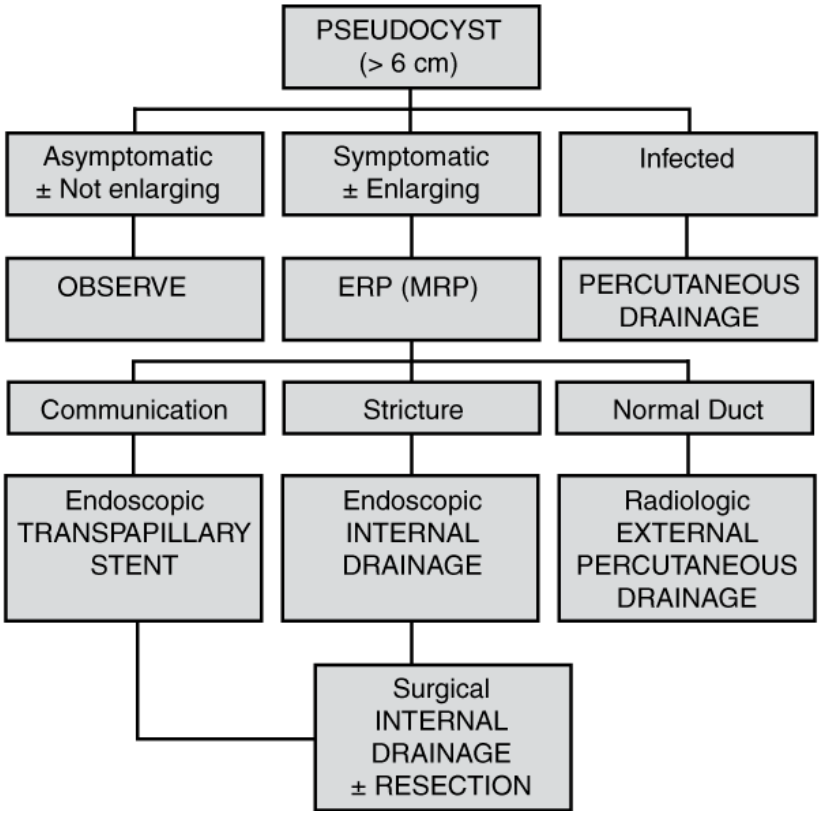
2. Rupture into peritoneum or pleural cavity.

3. Infection.

4. Thrombosis of splenic vein/ portal vein.

5. Haemorrhage from splenic artery.

Diagnosis and Management



In the absence of symptoms or evidence of enlargement, expectant management usually is reasonable. An enlarging asymptomatic pseudocyst that has been present for 6 weeks usually is treated. This relatively conservative approach is based on the low risk of complications. A natural-history study from India indicates that asymptomatic pseudocysts less than 7.5 cm in diameter

and without internal debris will resolve spontaneously at an average of 5 months.

There are two important rules in the treatment of pseudocysts. The first is that a cystic neoplasm must not be treated as a pseudocyst. The second is that elective external drainage of a pseudocyst must not be done if there is downstream and unrelieved pancreatic ductal obstruction because of the high risk of an external pancreatic fistula.

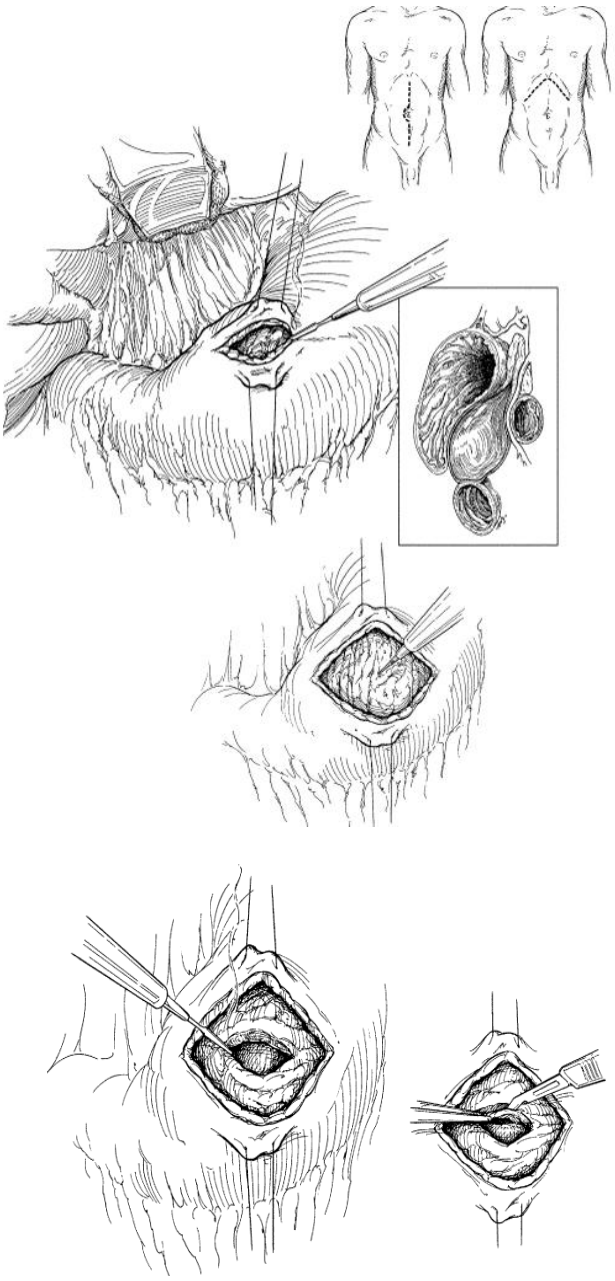
The approach to treatment depends on the nature of the pseudocyst, the pancreatic duct, and the fitness of the patient.

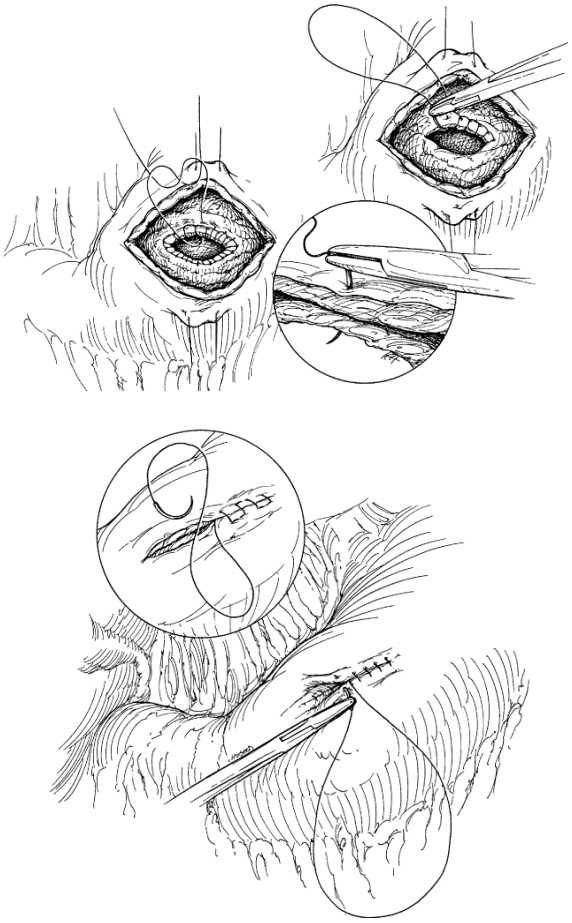
The following features of a pseudocyst are important in considering the most appropriate treatment:

- The thickness of the pseudocyst wall, which is usually a function of the duration of the pseudocyst. This is important because the operative drainage of a pseudocyst requires that it safely accept sutures or staples. After 4–6 weeks, this will not be an issue.
- The location of the pseudocyst. If adherent to the stomach or duodenum, the options are different than if the pseudocyst is deep within the retroperitoneum and covered by bowel loops.
- The contents of the pseudocyst. Blood may require prior embolization of a pseudoanuerysm. Pus will require drainage, percutaneous or open. Infected necrosum will require débridement.

- The pancreas and the pancreatic duct need separate consideration in planning the treatment of a pseudocyst. The pancreas may warrant treatment in its own right, especially if there is a ductal stricture, a dilated duct, or regional disease warranting resection. (6)

Open Surgical Treatment





A D'Egidio type II pseudocyst is best treated by internal drainage or resection. When there is a mature wall, internal drainage is the best surgical option. Recurrence rates should be less than 5%, and mortality should be less than 2%.

A pseudo cyst adherent to the posterior wall of stomach causing pressure symptoms is best treated by cysto gastrostomy. Those arising from body or tail of pancreas are treated by roux en Y cysto jejunostomy. In chronic pancreatitis a lateral pancreatico jejunostomy is done. Transduodenal drainage is done occasionally if the pseudo cyst impinges on the medial wall of duodenum. External drainage is done in critically ill patients, immature pseudo cysts with bleeding where under running has been done. External drainage with a trans

papillary stent of the duct along with long acting stomatostatin analogue is a good option in such conditions.

Radiologic Treatment

Percutaneous drainage is best suited to D'Egidio type I pseudo cysts in which there is no significant underlying duct abnormality or communication between the duct and pseudo cyst. In the setting of acute pancreatitis, catheter drainage is severely hampered by the inability to allow the ready egress of necrotic and viscous material. In the setting of chronic pancreatitis, the downstream obstruction of the duct gives rise to a high recurrence rate and/or an external fistula along the catheter tract. In simple, uncomplicated pseudo cysts, percutaneous drainage usually is successful, although this is the group with the fewest symptoms, the lowest complication rate, and the best chance of spontaneous resolution. (6)

The introduction of a transgastric approach to percutaneous drainage has almost abolished the problem of external pancreatic fistulas. This produces a percutaneous cystogastrostomy but requires an initial period of external transgastric drainage, clamping at 3 days, and then internalization at 2 weeks. Internalization can be helped with a concurrent endoscopic view, especially in the deployment of double pigtail catheters. The endoscopic approach is also used for the subsequent removal of the catheters. (6)

MATERIALS AND METHODS

This is a prospective study of 50 patients admitted with features of Acute Pancreatitis in K.A.P.V. Govt. Medical college Hospital, Trichy from September 2011 to November 2013. This study includes the patients of Trichy district and adjacent districts of Karur, Dindigul and Namakkal who were admitted for acute pancreatitis.

A proforma is attached to all the case sheets who were diagnosed as having Acute Pancreatitis. Serum amylase is taken as a routine in all suspected cases. All the data were entered in the proforma and the patients were followed till discharge or death.

The details of signs and symptoms, diagnosis, complications and outcome of the patient were entered in the proforma and they were tabulated and analysed. All the data obtained was entered in the master chart and tabulated for comparison and reference.

The observations were compared with the recent studies and literature available and conclusions were drawn.

MEATHOD OF COLLECTION OF DATA

Patients with acute onset of epigastric pain and vomiting who are suspected to have acute pancreatitis underwent serum amylase test. Those patients with elevated levels of serum amylase of more than 1000 IU underwent plain or contrast CT ABDOMEN on 3rd day. Simultaneously blood

investigations namely complete blood count, liver function test, renal function test, blood sugar, serum electrolytes and arterial blood gas analysis were done on the first day. The patients were graded according to Balthazar grading and modified CT severity index. The patients were given a score according APACHE II scoring system. Based on APACHE II scoring and CT grading, the severity, complications and prognosis of the patients was assessed.

INVESTIGATIONS

Hb%

Total count

Differential count

ESR

Blood urea

Serum cretinine

Sugar

sr. Amylase

sr. Electrolytes

Total bilurubin direct bilurubin AST ALP sr. Proteins

Chest x ray abdomen x ray erect AP view

Ultrasound abdomen

CT score

A – NORMAL PANCREAS

B – ENLARGEMENT OF PANCREAS – 2 points

C – INFLAMATORY CHANGES IN PANCREAS AND PERIPANCREATIC FAT – 2 points

D – ILLDEFINED SINGLE FLUID COLLECTION – 4 points

E – 2 OR MORE POORLY DEFINED FLUID COLLECTION – 4 points

PANCREATIC NECROSIS

NONE - 0

LESS THAN 30% - 2

30 to 50% - 4

MORE THAN 50% - 6

EXTRA PANCREATIC COMPLICATIONS – 2

APACHE II SCORE

a: acute physiology score (12	high abnormal rage					low abnormal range			
Physiological Variables	+4	+3	+2	+1	0	+	+2	+	+4
Temperature – rectal (°C)	≥41	39-40.9		38.5–	36–38.4	34–	32–33.9	30–	≤29.0
Mean arterial pressure (mm hg)	≥160	130–159	110–129		70–109		50–69		≤49
heart rate-ventricular response	≥180	140–179	110–139		70–109		55–69	40–	≤39
Respiratory rate non ventilated	≥50	35–49		25–34	12–24	10–	6–9		≤5
Oxygen: A – a DO or PaO (mm hg) $FiO_2 \geq 0.5$	≥500	350-499	200–349		<200 PO	PO 61–70		PO 55–60	PO <55
Arterial ph	≥7.7	7.6–7.69		7.5-7.59	7.33–		7.25–	7.15–	<7.15
Serum hCO – only if no	≥52	41.5–1.9		32–40.9	23–31.9		18–21.9	15–	<15
Serum sodium (mmol/I)	180	160–179	155–159	50–154	130–		120–	111–	≤110
Serum potassium (mmol/I)	≥7	6–6.9		5.5–5.9	3.5–5.4	3–3.4	2.5–2.9		<2.5
Serum creatinine (umol/l)	≥350	200–340	150–190		60–140		<60		
haematocrit (%)	≥60		50–50.9	46–49.9	30–45.9		20–29.9		<20
White Blood cell court (x1000	≥40		20–39.9	15–19.9	30–14.9		1–2.9		<1
Glasgow Coma Score (GCS)	Score = 15 minus actual GCS								

B. age points					
age years	Points	History	Points for elective	Points for emergency	
≥ 44	0	Liver: Biopsy proven cirrhosis and documented portal hypertension or prior	2	5	A: APS score
45–54	2	Cardiovascular NYhA class IV	2	5	B: Age Points score
55–64	3	Respiratory eg. Severe COPD, hypercapnia, home O2 pulmonary hypertension	2	5	C: Chronic health points score
65–74	5	Renal chronic dialysis	2	5	
≥ 75	6	Immunocompromised	2	5	Total apache II

DEFINITIONS FOR ACUTE PANCREATITIS ACCORDING TO ATLANTA CLASSIFICATION[10]

Criteria of illness severity in acute pancreatitis

Local complications	Necrosis: focal or diffuse area of non viable pancreatic parenchyma, with necrosis of peripancreatic fat (> 30% of the gland or > 3 cm)
	Pseudocyst: pancreatic juice collection surrounded by a wall of granulation or fibrous tissue that is developed as a consequence of acute or chronic pancreatitis or pancreatic traumatism
	Abscess: pus collection well defined that has scarce or no amount of pancreatic necrosis
Systemic complications (organic failure)	Respiratory failure: PaO2 < 60 mmHg
	Shock: systolic BP < 90 mmHg
	Renal failure: creatinine > 2 mg/dL after rehydration
	Upper gastrointestinal bleeding: > 500 mL/24hrs
Bad prognosis data	Ranson's scale ≥ 3
	APACHE II scale ≥ 8

MANAGEMENT

Patients were broadly divided into two groups. Those who had pancreatic CT score <6 and APACHE II score was less than 8 were termed as mild pancreatitis. Patients with CT score> 6 and APACHE II score more than 8 were termed as severe pancreatitis and were admitted in intensive care unit. All patients of mild pancreatitis were managed conservatively. Fluid resuscitation at the rate of 150 – 200ml/hour using normal saline,dextrose normal saline and ringer lactate solution. Strict hourly urine input and output chart was maintained. Urine output was maintained to more than 0.5ml/kg/hour. Diuretics was used in patients with low output after measuring renal parameters, blood pressure and central venous pressure on ventilator patients. Antibiotics given include ceftriaxone 1g i.v b.d, amikacin 250mg i.v b.d, metronidazole 500mg i.v , piperecillin tazobactam 4.5g i.v b.d. Octeotride 100mg tds was used in severe pancreatitis patients. Serial blood investigations were done. Patients with hyperglycemia were started on regular insulin.

Those having random blood glucose of more than 250mg% were started on 5 units of actrapid insulin in 0.9% DNS solution. Diuretics were used in patients with elevated renal parameters. Hypokalemia less than 3.5 meq/l was corrected with intravenous infusion of potassium. 2 ampules each containing 20meq/l were given as a continuous infusion over 4 – 6 hours in normal saline through a peripheral line. Hypocalcemia of less than 8meq/l was corrected with 10% of 10ml calcium gluconate in 100ml normal saline over 10 – 20 minutes.

Patients with severe acute respiratory distress syndrome as evidenced by bilateral pulmonary crepitations, fluffy lung infiltrates (groung glass appearance) and hypoxia less than 90% were ventilated and were treated with antibiotics, hydrocortisone 100mg i.v tds, soda bicarbonate and chest physiotherapy. All patients were kept nil per oral for first few days due to abdominal pain, anorexia and vomiting. Those with mild pancreatitis were started on sips of water as soon as patient tolerated. Soft diet containing less fat was started the following day.

Mild pancreatic ascites, pancreatic pleural effusion and acute fluid collection in the lesser sac were managed conservatively and eventually subsided.

One patient developed massive ascites and did not resolve by the end of 3rd week. Pigtail catheter insertion was done and daily ascetic tapping was done not more than 1 litre. Patient eventually developed external pancreatic fistula as evidenced by high amylase content in ascetic fluid. The patient also had more than 5o% necrosis on contrast CT. Since the patient was symptomatic at the end of 4 weeks, an elective delayed pancreatic necrosectomy was done.

Another patient had infected pancreatic necrosis as evidenced by gas in CT abdomen and pancreatic necrosectomy was done. In both patients, abdomen was opened in midline. Ascitic fluid sucked out. The hepatic flexure and splenic flexure were mobilized and taken down. Duodenum was mobilized till inferior venecava. Gastrocolic omentum was opened and lesser sac entered. With gentle

finger dissection, all the necrosum was debrided from the body and tail of pancreas. Minimal oozing was controlled by packing and suturing the pancreatic tissue/ducts with silk. Two large bore abdominal drains were kept and abdomen closed in single layer. Post operatively patients were kept nil per oral and treated with intravenous fluids, antibiotics, octeotride, analgesics and proton pump inhibitors. Patients developed glucose intolerance and insulin was started. Oral fluids was started on 10^{th} post op day. Drainage tube removed on 12^{th} post op day.

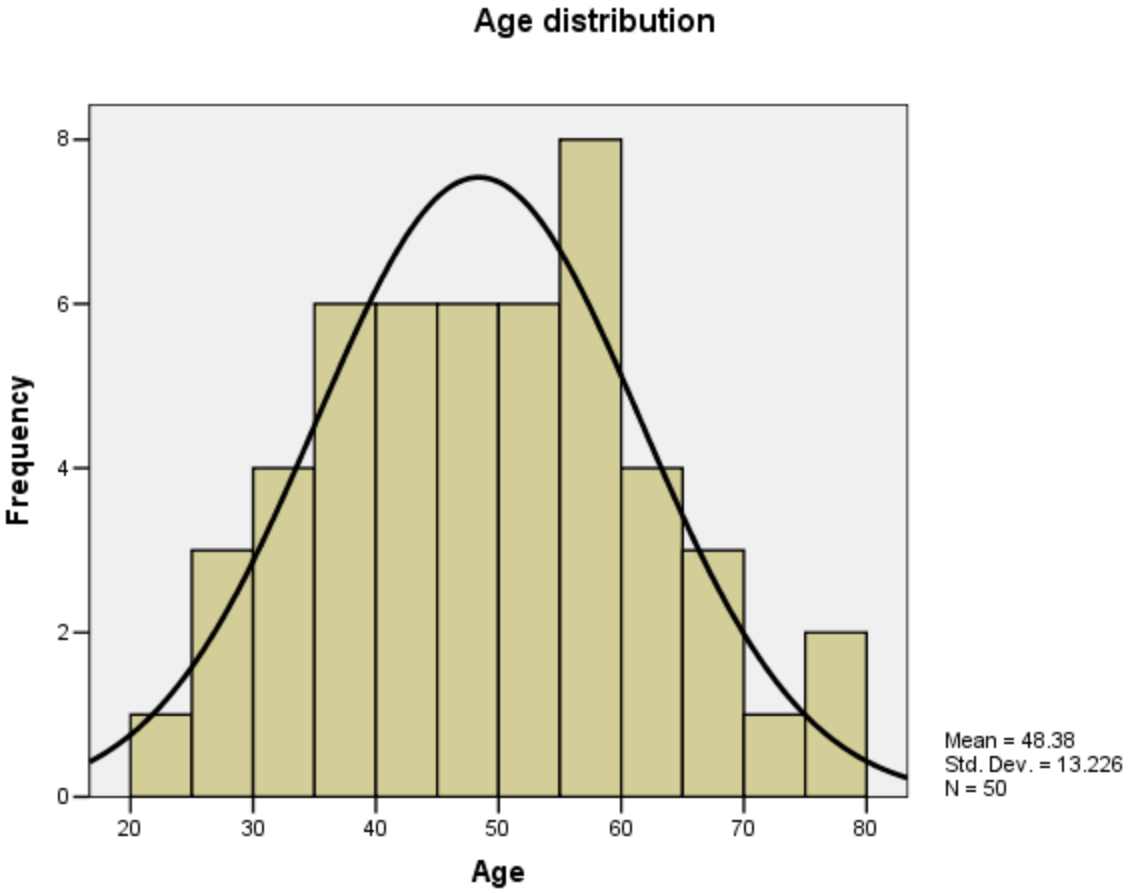
After collection of the necessary data, the mean age of presentation of pancreatitis, gender distribution was found. Ultrasound abdomen, chest x – ray findings, APACHE II score and CT score were tabulated. Complications during the treatment period were also assessed. A correlation between APACHE II score , CT score and the severity of acute pancreatitis was determined.

OBSERVATION

A total of 50 patients admitted during the period from September 2011 to November 2013 at K.A.P.V. Government Medical college Hospital were included in the study. After admission the data regarding the patient were entered into the proforma and followed up until discharge/death. The data of 50 patients were entered in the master chart and analysed. The data obtained were compared and the observations done were given below.

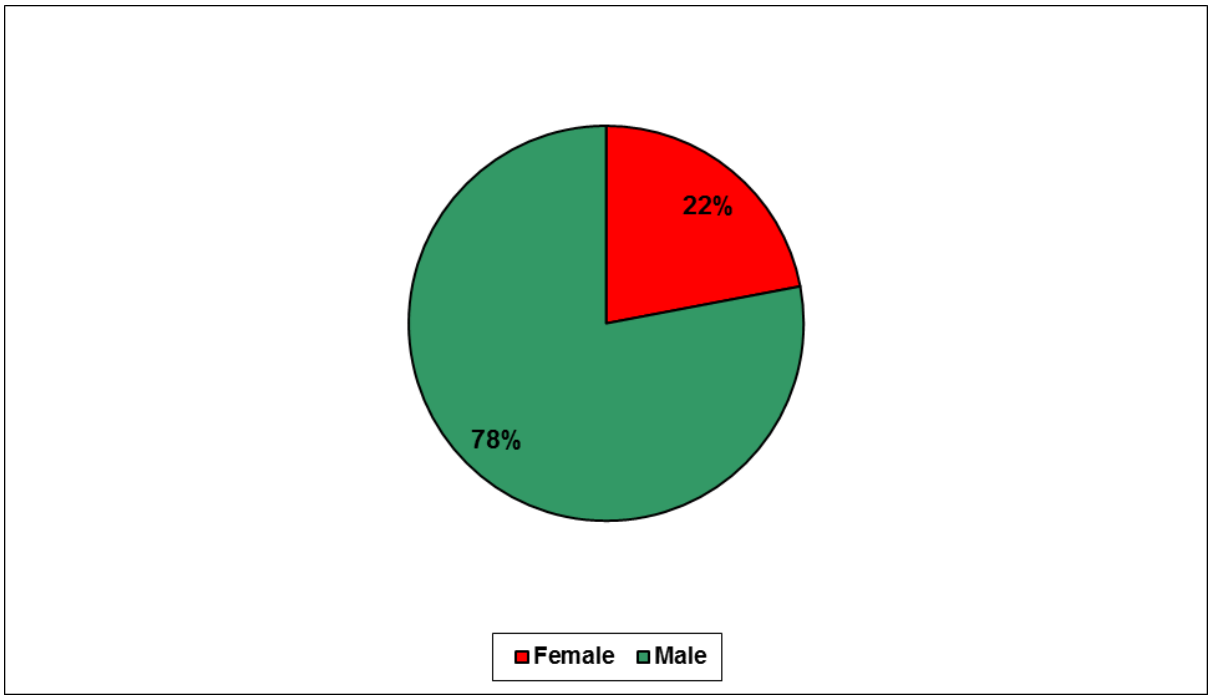
Age distribution:

	No. of patients	**Percent**
< 30	4	8
30 – 40	10	20
40 – 50	12	24
50 – 60	14	28
60 – 70	7	14
70 – 80	2	4
> 80	1	2
Total	50	100


Age distribution
Frequency
0
2
4
6
8
20
30
40
50
60
70
80
Age
Mean = 48.38
Std. Dev. = 13.226
N = 50

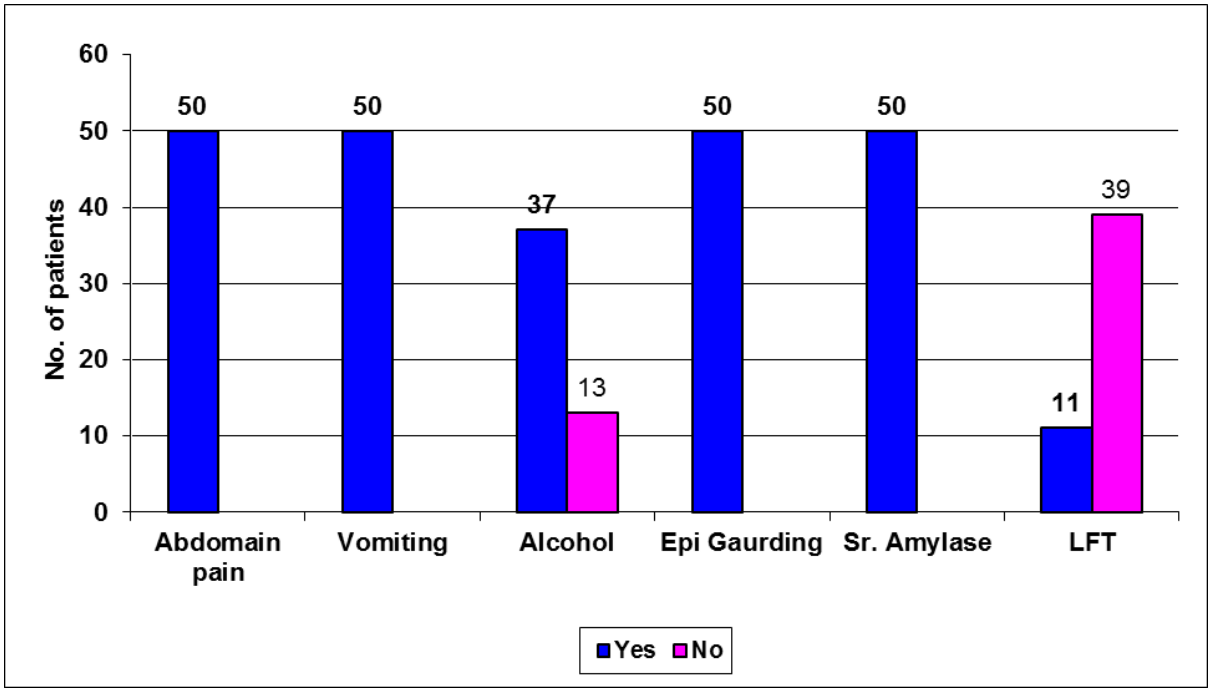
SEX DISTRIBUTION

Gender	**No. of patients**	**Percent**
Female	11	22
Male	39	78
Total	50	100



SIGNS AND SYMPTOMS:

	Yes (%)	**No (%)**
Abdomain pain	50(100)	
Vomiting	50(100)	
Alcohol	37(74)	13(26)
Epi Gaurding	50(100)	
Sr. Amylase	50(100)	
LFT	11(22)	39(78)



CXR

	No. of patients(%)
Normal	45(90)
Abnormal (pleural effusion, fluffy infiltrates)	5(10)

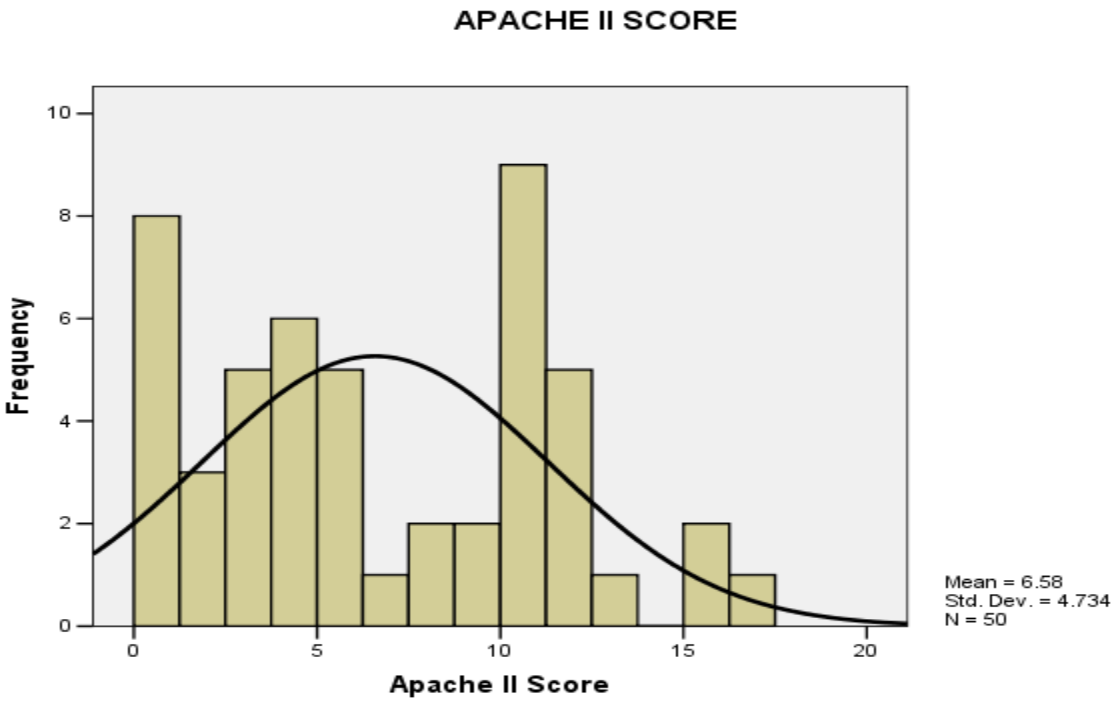
USG

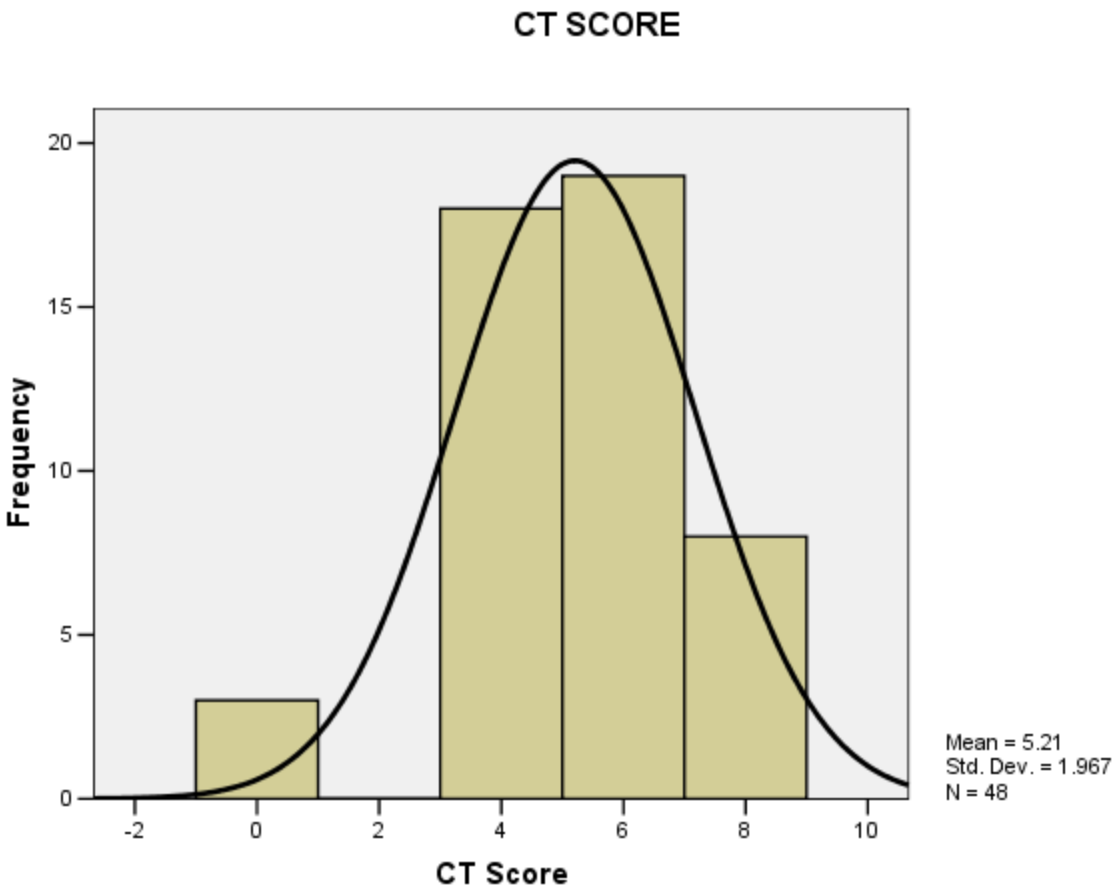
	No. of patients(%)
Diffusely enlarged pancreas	34 (68)
Diffusely enlarged pancreas,gallstone +	5 (10)
Enlarged pancreas	1 (2)
Pancreas not visualised	10 (20)
Total	50 (100)

Statistics

	Age	Apache II Score	CT Score
No. of patients	50	50	48
Missing values	0	0	2
Mean	48.38	6.58	5.21
Median	48.5	5.5	6
Mode	55	0	6
Std. Deviation	13.23	4.73	1.97
Variance	174.93	22.41	3.87
Skewness	0.17	0.29	-0.79
Std. Error of Skewness	0.34	0.34	0.34
Kurtosis	-0.42	-0.97	1.22
Std. Error of Kurtosis	0.66	0.66	0.67

a. Multiple modes exist. The smallest value is shown




CT SCORE
Frequency
20
15
10
5
0
-2
0
2
4
6
8
10
CT Score
Mean = 5.21
Std. Dev. = 1.967
N = 48

COMPLICATIONS:

C1. ACUTE FLUID COLLECTION

C2. VENOUS THROMBOSIS

C3. ACUTE RESPIRATORY DISTRESS SYNDROME

C4. PANCREATIC ASCITES/ EFFUSION

C5. PARALYTIC ILEUS

C6. ORGAN FAILURE/ MULTIORGAN DYSFUNCTION SYNDROME

C7. PANCREATIC NECROSIS

C8. INTESTINAL OBSTRUCTION

C9. HYPOCALCEMIA/HYPERGLYCEMIA

C10. PSUEDOCYST

C11. INTESTINAL PERFORATION

C12. DIC/ ENCEPAHLOPATHY

Complications

	Presence (%)	**Absence (%)**
C1	12(24%)	38(76%)
C2		50(100%)
C3	5(10%)	45(90%)
C4	5(10%)	45(90%)
C5	35(70%)	15(30%)
C6	5(10%)	45(90%)
C7	46(92%)	4(8%)
C8		50(100%)
C9	10(20%)	40(80%)

Statistical tests used : Chi-Square test and Fisher's exact test

Null hypothesis : There is no association between APACHE II Score and Complication

Alternative hypothesis: There is a association between APACHE II Score and Complication

Complications		Apache II Score		Total	P Value	Result
		< 8	> 8			
c1	Absence	30	8	38	<0.001	Highly Significant
	Presence	0	12	12		
c3	Absence	30	15	45	0.007	Significant
	Presence	0	5	5		
c4	Absence	30	15	45	0.007	Significant
	Presence	0	5	5		
c5	Absence	3	12	15	< 0.001	Highly Significant
	Presence	27	8	35		
c6	Absence	30	15	45	0.007	Significant
	Presence	0	5	5		
c7	Absence	3	1	4	0.641	Not Significant
	Presence	27	19	46		
c9	Absence	30	10	40	<0.001	Highly Significant
	Presence	0	10	10		
Total		30	20	50		

Inference:

The above table shows that there is a association between APACHE II Score >8 and Complication c1,c3,c4,c5,c6 and c9 and there is no association between APACHE II Score and complication c7.

Statistical tests used : Chi-Square test and Fisher's exact test

Null hypothesis : There is no association between CT Score and Complication

Alternative hypothesis: There is a association between CT Score and Complication

Complications		CT Score		Total	P Value	Result
		< 6	> 6			
c1	Absence	33	5	38	0.046	Significant
	Presence	7	5	12		
c3	Absence	39	6	45	0.004	Highly Significant
	Presence	1	4	5		
c4	Absence	37	8	45	0.258	Not Significant
	Presence	3	2	5		
c5	Absence	13	2	15	0.44	Not Significant
	Presence	27	8	35		
c6	Absence	40	5	45	<0.001	Highly Significant
	Presence	0	5	5		
c7	Absence	4	0	4	0.571	Not Significant
	Presence	36	10	46		
c9	Absence	37	3	40	<0.001	Highly Significant
	Presence	3	7	10		
Total		40	10	50		

Inference:

The above table shows that there is a association between CT Score >6 and Complication c1,c3,c6 and c9 and there is no association between CT Score and complication c4,c5 and c7.

DISCUSSION

A total of 50 patients admitted during the period from September 2011 to November 2013 at K.A.P.V. Government Medical college Hospital were included in the study. After admission the data regarding the patient were entered into the proforma and followed up until discharge/death. The data of 50 patients were entered in the master chart and analysed.

Average age of presentation of acute pancreatitis in our study was 48 years.

Acute pancreatitis due to alcohol abuse was more common in age group between 50 - 60 years.

There is male pre ponderance in patients with acute pancreatitis

The Male to Female ratio is almost 4: 1

All the patients with acute pancreatitis had upper abdominal pain and vomiting.

Alcohol abuse was the common cause of acute pancreatitis which accounts for 90% of the cases followed by gall stone disease which accounts for 10% of the cases.

Serum amylase was the first line of investigation in all cases. All cases showed hyperamylasia with serum amylase levels of more than 1000 IU. Liver function test was normal in 45 patients. Only 5 patients with gall stone disease showed elevated total bilirubin, direct bilirubin and alkaline phosphatase levels. Complete blood count showed elevated white blood cells in all patients. 5

patients showed elevated haematocrit of more than 44%. 10 patients had blood sugar of more than 250mg%. 10 patients had elevated renal parameters . 10 patients had hyponatremia (sodium less than 135meq/l) and hypokalemia (potassium less than 3.5meq/l) due to severe vomiting. Arterial blood gas analysis was done and it showed that 10 patients had metabolic acidosis as evidenced by decreased pH and decreased bicarbonate. 8 patients had hypocalcemia with calcium levels of less than 8 meq/l. 5 patients had signs of Acute respiratory distress syndrome as evidenced by fluffy lung infiltrates on chest x ray, saturation of less than 80% and partial pressure of oxygen of less than 60 mm Hg. Glasgow coma scale was 15/15 in all patients.

Chest infiltrates were present in 5 patients. Pleural effusion was present in 5 patients

Ultra sound abdomen

Diffusely enlarged and hypoechoic gland was present in 30 patients. Gall stone was present in 5 patients. There were no stones in the common bile duct at the time of presentation. Extra pancreatic fluid collections (lesser sac, anterior Para renal space) were present in 5 patients. 10 patients had dilated bowel loops and hence could not be assessed properly with ultrasound abdomen. Routine blood investigations and arterial blood gas analysis was done on the day of admission for all patients and APACHE II score was calculated. We found in our study that the mean APACHE score was 6.58, the standard deviation is 4.73.

CT SCORE: 30 patients had GRADE C i.e. inflammatory changes in pancreas and peripancreatic fat. 5 patients had gall stone disease.5 patients had GRADE D i.e., ill-defined single fluid collection. 5 patients had GRADE E i.e. 2 or more poorly defined fluid collection. 30 patients had necrosis of less than 30%.10 patients had necrosis of 30 to 50%. 10 patients had necrosis of more than 50%. The mean CT score was found to be 5.21 and the standard deviation was 1.97. Patients were assessed in the ward for the complications and it was found that paralytic ileus, acute fluid collection, hyperglycemia, hypocalcemia, hypokalemia were common.

Based on all the above information, a corerelation between acute pancreatitis, APACHE II score and CT score was established. We found that there is a significant correlation between APACHE score > 8, CT score > 6 and complications namely paralytic ileus, metabolic disturbances, acute fluid collection, pancreatic ascites, pancreatic effusion and acute respiratory distress syndrome.

CONCLUSION

In our study, the main etiologic factor was found to be alcohol. Acute pancreatitis is common in young and middle aged males. Mild pancreatitis as evidenced by CT score < 6and APACHE II score < 8 have better prognosis.. Oral diet is tolerated faster, sepsis is minimal and local complications are lesser. Severe pancreatitis as evidenced by CT scores > 6 and APACHE II score of > 8 have severe metabolic and electrolyte disturbances. Prognosis is guarded specially those who have acute respiratory distress syndrome and features of multi organ dysfunction syndrome. Acute fluid collection, pancreatic ascites, pancreatic effusion eventually subsided on their own. External pancreatic fistula, organized pancreatic necrosis and infected pancreatic necrosis were taken up for surgery (pancreatic necrosectomy and debridement of body and tail respectively).

There is a significant correlation between APACHE score > 8, CT score > 6 and complications namely paralytic ileus, metabolic disturbances, acute fluid collection, pancreatic ascites, pancreatic effusion and acute respiratory distress syndrome.

REVIEW OF RECENT LITERATURE

The following studies are comparable to our study.

1. Acute Pancreatitis: Assessment of Severity with Clinical and CT Evaluation; Emil J. Balthazar, MD.
2. The clinical prognostic indicators of Acute pancreatitis by APACHE II scoring; Dr. Rithin suvarna; AJ Institute of Medical Sciences; Mangalore; India. (13)
3. World journal of Acute Pancreatitis update; Dulce M Santamaria ; Carlos Taxonera; Maneul Giner.(14)
4. Elmer press; Gastroenterology research; predicting acute pancreatitis severity: comparision of prognostic scores; Marco Simoes ; Patricia Alves

In all the above studies, the correlation between Acute Pancreatitis, CT grading and APACHE scoring were studied. It was found that patients with CT score of more than 6 or more than 30% necrosis and APACHE score of more than 8 had more complications and had poor prognosis. Patients with APACHE II score of less than 8 had fewer complications and good prognosis.

RECENT ADVANCES IN PANCREATITIS[11]

Enteral Nutrition in Acute Pancreatitis (11):

Usually patients with acute pancreatitis are kept nil per oral. The rationale behind this is to give rest to pancreas. Also the patients are anorectic and have severe vomiting. This concept of putting pancreas to rest was developed by Ragins. However it was in canines. Pancreas secretes protein enzymes, fluid volume and bicarbonates. Of this protein enzymes are responsible for auto digestion of gland. Hence suppression of only protein enzymes will be sufficient to halt the disease process. Pancreatic secretion is stimulated by three levels. Cephalic phase mediated through vagus, gastric phase mediated through gastrin and intestinal phase mediated through vagus, CCK, VIP and secretin like hormone. Pancreatic secretion reaches maximum when food is taken in mixed form of 20kcal/lg containing more of fat. Pancreatic secretion is significantly reduced when food has bypassed stomach and duodenum and is directly given around 60cms from ligament of treitz (bypassing all three phases of pancreatic stimulation). Also, elemental feeds cause less stimulation as they contain less fat. The free fatty acids combine with trypsin and levels of trypsin is reduced. All patients must receive enteral nutrition as it preserves intestinal mucosal integrity and chances of infection are less.

Although the use of glutamine supplementation, immunonutrition and prebiotics, and/or probiotics is conceptually sound and attractive, their use is not

supported by large-scale studies .The precise timing for enteral nutrition is not addressed but early enteral nutrtion within 36 hours is beneficial. The chances of pancreatic infection, multi organ failure are less. Enteral nutrition is best provided by placement of nasojejunal tubes either endoscopically or ragraphically. Prolonged feeding for 4 – 6 weeks may need laproscopic placement of jejunal tubes. Feeds can be given in bolus or continuous infusion. Generally, for mild AP it is recommend to initiate EN if patients cannot consume normal food after 5–7 days. For severe AP nutritional support is indicated when it becomes evident that the patient will not be able to tolerate oral intake for a prolonged period of time, for example, for at least 7 days. This assessment can usually be made within the first 3-4 days of admission. EN should be supplemented by parenteral nutrition if needed. Also, in severe pancreatitis with complications such as pancreatic fistulas, ascites, and pseudocysts, tube feeding can be given uneventfully. If gastric feeding is not tolerated, the jejunal route should be tried and continuous feeding in stead of bolus feeding should be used. In gastric outlet obstruction, feeding beyond the obstruction with the tube tip distal to the obstruction should be tried. If this is impossible, parenteral nutrition should be given. In case of surgery for complications of AP, an intraoperative jejunostomy for postoperative feeding is feasible.

SCORING SYSTEM[12]

A recent development in scoring system in acute pancreatitis during the first 24 hours is Bedside Index of Severity in Acute Pancreatitis (BISAP).

1 point for each criteria:

1. Blood urea nitrogen level more than 8.9mmol/l
2. Impaired mental status
3. Systemic inflammatory response syndrome
4. Age > 60 years
5. Pleural effusion on radiography.

A score of more than 3 indicates increased risk of death.

Criteria for systemic inflammatory syndrome:

1. heart rate > 90/min
2. respiratory rate more than 20/ min or partial pressure of carbondioxide <32mm Hg.
3. Body temperature <36 or >38leucocyte count < 4000 or >12000/ cumm or >10% immature neutrophils (bands).

PROFORMA

Name: Age: Sex:

I.P No.: Ward:

Date of admission:

Date of discharge:

I PRESENTING FEATURES

1. Abdominal pain
2. Vomiting
3. Fever
4. Any other

II. PAST HISTORY

H/O JAUNDICE

H/O GALL STONES

H/O SURGERIES/ ERCP

III. MENSTRUAL AND OBSTETRIC HISTORY

IV. FAMILY HISTORY

V.PERSONAL HISTORY

V. GENERAL EXAMINATION

BUILT & NOURISHMENT

PULSE RATE

BLOOD PRESSURE MEAN ARTERIAL PRESSURE

RESPIRATORY RATE

TEMPERATURE

GCS

SIGNS OF DEHYDRATION

ANAEMIA

JAUNDICE

CYANOSIS

CLUBBING

LYMPHADENOPATHY

PEDAL OEDEMA

VI. EXAMINATION OF ABDOMEN

1. EPIGASTRIC TENDERNESS
2. GAURDING
3. RIGIDITY
4. GREY TURNER SIGN, CULLEN'S SIGN, FOX SIGN
5. ASCITES
6. PER RECTAL EXAMINATION –
7. PER VAGINAL EXAMINATION –

VII. EXAMINATION OF OTHER SYSTEMS

RESPIRATORY SYSTEM – PLEURAL EFFUSION/ CONSOLIDATION

CARDIOVASCULAR SYSTEM

CENTRAL NERVOUS SYSTEM – GCS -

VIII. INVESTIGATIONS

Hb%

Total count

Differential count

ESR

Blood Urea

Blood sugar

Serum creatinine

Serum electrolytes

Serum amylase

Total bilurubin

Direct bilurubin Indirect bilurubin

AST ALT ALP

Serum proteins

X-Ray abdomen erect

Chest X-Ray PA view

Ultrasonogram abdomen

PLAIN/ CONTRAST CT FINDING - BALTHAZAR SCORING –

A – NORMAL PANCREAS

B – ENLARGEMENT OF PANCREAS

C – INFLAMATORY CHANGES IN PANCREAS AND PERIPANCREATIC FAT

D – ILLDEFINED SINGLE FLUID COLLECTION

E – 2 OR MORE POORLY DEFINED FLUID COLLECTION

PANCREATIC NECROSIS

NONE - 0

LESS THAN 30% - 2

30 to 50% - 4

MORE THAN 50% - 6

EXTRA PANCREATIC COMPLICATIONS – 2

BALTHAZAR SCORING/ MODIFIED CT SEVERITY INDEX-

APACHE II SCORING

PARAMETER	SCORE
Temperature	
Mean arterial pressure	
Heart rate	
Respiratory rate	
A-aPo2	
PAo2	
Ph	
Bicarbonate	
Sodium	

Potassium	
Creatinine	
PCV	
WBC	
GCS	
Age adjustment	
Chronic health adjustment	
Total	

BASED ON APACHEII SCORING AND CT GRADING:

1. MILD PANCREATITIS
2. SEVERE PANCREATITIS

MANAGEMENT

1. FLUID RESUCITATION
2. ELECTROLYTE CORRECTION
3. STOMACH DECOMPRESSION
4. ANTIBIOTICS
5. ANALGESICS
6. OCTEOTRIDE
7. CALCIUM GLUCONATE
8. HOURLY INPUT OUTPUT
9. TOTAL INPUT OUTPUT

COMPLICATIONS

LOCAL	REGIONAL	SYSTEMIC
ACUTE FLUID COLLECTION	VENOUS THROMBOSIS	ACUTE RESPIRATORY DISTRESS SYNDROME
PANCREATIC ASCITES/ EFFUSION	PARALYTIC ILEUS	ORGAN FAILURE/ MULTIORGAN DYSFUNCTION SYNDROME
PANCREATIC NECROSIS	INTESTINAL OBSTRUCTION	HYPOCALCEMIA/HYPERGLYCEMIA
PSUEDOCYST	INTESTINAL PERFORATION	DIC/ ENCEPAHLOPATHY

OUTCOME OF THE PATIENT DURING HOSPITAL STAY:

CONSENT

ஒப்புதல் படிவம்

தலைப்பு:

Acute Pancreatitis (கணையப் புண்)ன் வீரியத்தை, மருத்துவப் பரிசோதனைகள் மூலம் ஆராய்து அறிதல்.

பங்குபெறுபவர் பெயர் :
பரிசோதனை செய்யும் இடம் :
பரிசோதனை எண் :
நோயாளியின் எண் :

1.நான் இப்பரிசோதனையின் தகவல் படிவம் .....தேதியிட்ட படிவததை படித்து புரிந்துக் கொண்டேன் என உறுதியளிக்கிறேன். அதில் உள்ள சந்தேகங்களை நிவர்த்தி செய்யவும் வாய்ப்பு அளிக்கப்பட்டேன்.

2.என்னுடைய பங்களிப்பு சுய விருப்பத்தின் பேரில் தான் என்பதையும், இதில் இருந்து எந்த நிலையிலும் காரணம் தெரிவிக்காமல் விளகிக்கொள்ளவும் எனக்கு உரிமையுள்ளதையும் அறிந்துக் கொண்டேன். இது என்னுடைய சி மருத்துவ சிகிச்சையை எந்த விதததிலும் பாதிப்பு ஏற்படுத்தாது என உயர்ந்துக் கொண்டேன்.

3.என்னுடைய பரிசோதனை முடிவுகளை எப்பொழுது வேண்டுமானாலும் பயன்படுத்திக்கொள்ள இச்சோதனை அதிகாரிகளுக்கு முழு உரிமை அளிக்கிறேன்.

4.இதன் மூலம் நான் இச்சோதனையில் பங்குபெற முழு சம்மதம் அளிக்கிறேன்.

1)நோயாளியின் கையெப்பம்

2)நோயாளியின் உறவினர் கையொப்பம்

3)பரிசோதகரின் கையொப்பம்

தாங்களுக்கு (ACUTE PANCREATITIS) கணையத்தில் புண்ணாகியுள்ளதால் நாங்கள்ழி உங்களிடம் சில கேள்விகள், இரத்தப்பரிசோதனை, USG Scan மற்றும் CT-Scan ஆகியவை செய்து தங்கள் நோயின் தீவிரத்தை அறிவதற்கான சோதனையில் தங்களை இனைத்துக் கொள்வதற்கான ஒப்புதல் படிவம் இந்த சோதனையில் 50 நோயாளிகள் பங்கேற்பார்கள். இந்த சோதனையில் இணைத்துக் கொள்வதற்கும், எந்த நிலையில் வேண்டுமென்றாலும் வெளியேறுவதற்கும் தங்களுக்கு முழு சுதந்திரம் அளிக்கப்படும். இச்சோதனையில் இணைத்துக் கொள்வதனாலும், இல்லையென்றாலும் சிகிச்சையில் தங்களுக்கு எந்தவித மாறுதல்களும் இல்லை என உறுதியளிக்கிறோம். மேலும் இதில் தங்களை இணைத்துக்கொள்வதின் மூலம் தாங்கள் பின்வரும் சந்ததியினருக்கு மேற்சொன்ன நோயின் அறிகுறிகளைக் கொண்டு மேலும் சிறந்த முறையில் சிகிச்சையளிக்கும் வாய்ப்பை ஏற்படுத்திக் கொடுக்கிறீர்கள். இதில் இணைவதனால் தாங்கள் இரண்டு வாரங்கள் வரை மருத்துவமனையில் தங்க நேரிடலாம். இதனால் தங்களுக்கு ஏற்படும் சிரமங்களான இரத்தக்கட்டி (Hematoma at arterial Puncture), ஒவ்வாமை (Anaphylaxis for contrast) ஆகியவை ஏற்பட வாய்ப்பு உள்ளது என்பதையும் அதற்கான சிகிச்சைகள் இம்மருத்துவமனையில் உள்ளது என்பதையும் தங்களுக்கு தெரிவித்துக் கொள்கிறோம். இதன் மூலம் பெறப்படும் அனைத்து தகவல்களும் மிகவும் பாதுகாப்பாக வைக்கப்படும் என உறுதியளிக்கிறோம். இச்சோதனை எங்கள் ஆசிரியர்கள் மற்றும் பேராசிரியர்களால் கண்காணிக்கப்படும். இதில் பங்கேற்பதற்கு எங்களுடைய மனப்பூர்வமான நன்றியை தங்களுக்கு தெரிவித்துக்கொள்கிறோம்.

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15. Elmer press; Gastroenterology research; predicting acute pancreatitis severity: comparision of prognostic scores; Marco Simoes ; Patricia Alves

SL NO	NAME	AGE/SEX	ABD PAIN	VOMITING	ALCOHOL	EPI GAURDING	SR AMYLASE	LFT	CXR	USG	APACHE II	CT SCORE	COMPICATION
1	Kuppusamy	58/m	y	Y	y	y	y	n	Fluffy lung infiltrates	Diffusely enlarged pancreas	17	8	1,3,4,5,6,7,9
2	Kumarasamy	48/M	y	Y	y	y	y	y	N	Pancreas not visualised	12	6	1,4,7
3	Pitchai	61/m	y	Y	y	y	y	n	N	Diffusely enlarged pancreas	11	6	7,9
4	Senthilkumar	70/m	y	Y	y	y	y	n	N	Diffusely enlarged pancreas	11	6	7,9
5	Kumar	49/m	y	Y	y	y	Y	y	N	Pancreas not visualised	10	6	3,5,,7,
6	Thiyagarajan	46/m	y	Y	y	y	Y	n	N	Pancreas not visualised	12	6	1,4,7
7	Kolandaivel	68/m	y	Y	y	y	Y	n	N	Diffusely enlarged pancreas	10	6	1,4,7
8	Vetrivel	47/m	y	Y	y	y	Y	n	N	Pancreas not visualised	10	6	1, 7
9	Hemalatha	56/f	y	Y	n	y	Y	y	N	Diffusely enlarged pancreas,gallstone +	13	8	1, 7, 9
10	Tamilselvi	44/f	y	Y	n	y	Y	n	N	pancreas not visualised	10	6	7,9
11	Velmurugan	45/m	y	Y	y	y	Y	y	N	Pancreas not visualised	10	6	1, 7
12	Kalaiarasi	50/f	y	Y	n	y	Y	n	N	Pancreas not visualised	6	6	NIL
13	Prabhu	75/m	y	Y	y	y	Y	n	N	Diffusely enlarged pancreas	8	6	NIL
14	Palanisamy	60/m	y	Y	y	y	Y	n	N	Diffusely enlarged pancreas	6	2	5,7
15	Perumal	80/m	y	Y	y	y	Y	n	N	Diffusely enlarged pancreas	9	6	1, 7
16	Sudha	24/f	y	Y	n	y	Y	y	N	Diffusely enlarged pancreas	1	6	5,7
17	Ravikumar	36/m	y	y	y	y	Y	n	N	Diffusely enlarged pancreas	5	4	5,7

18	Thangaraj	37/m	y	y	y	y	Y	n	N	Diffusely enlarged pancreas	4	4	5,7
19	Periyammal	39/f	y	y	N	y	Y	n	N	Diffusely enlarged pancreas	4	4	5,7
20	Kandhasamy	38/m	y	y	y	y	Y	n	N	Diffusely enlarged pancreas	4	4	5,7
21	Sivakumar	40/m	y	y	y	y	Y	n	N	Diffusely enlarged pancreas	4	4	5,7
22	Nagur pitchai	28/m	y	y	n	y	Y	y	N	Diffusely enlarged pancreas,gallstones+	3	6	5,7
23	Premalatha	43/f	y	y	n	y	Y	n	N	pancreas not visualised	3	4	5,7
24	Shanthi	26/f	y	y	n	y	Y	y	N	Diffusely enlarged pancreas,gallstones+	0	6	5,7
25	Jayaraj	35/m	y	Y	y	y	Y	n	N	Diffusely enlarged pancreas	0	4	5,7
26	Kaderesan	33/m	y	Y	y	y	Y	n	N	Diffusely enlarged pancreas	0	4	5,7
27	Aiyappan	31/m	y	Y	y	y	Y	n	N	Diffusely enlarged pancreas	3	4	5,7
28	Vivek	34/m	y	Y	y	y	Y	n	N	Diffusely enlarged pancreas	0	4	5,7
29	Pushpavalli	41/f	y	Y	n	y	Y	n	N	Pancreas not visualised	4	4	5,7
30	Vinoth	32/m	y	Y	y	y	Y	N	N	Diffusely enlarged pancreas	0	4	5,7
31	Kannaghi	57/f	y	Y	n	y	Y	Y	N	Diffusely enlarged pancreas,gallstones+	7	8	5,7
32	Ganeshan	58/m	y	Y	y	y	Y	N	N	Diffusely enlarged pancreas	3	0	5,7
33	Parimala	42/f	y	Y	n	y	Y	N	N	Pancreas not visualised	1	4	5,7
34	Ramesh	55/m	y	Y	y	y	Y	N	N	Diffusely enlarged pancreas	2	4	5,7
35	Meena	54/f	y	Y	n	y	Y	N	N	Diffusely enlarged pancreas	10	2	1 , 7, 9
36	Suresh	51/m	y	Y	y	y	Y	N	N	Diffusely enlarged pancreas	5	6	5,7
37	Subramani	52/m	y	Y	N	y	Y	N	N	Diffusely enlarged pancreas,gallstone+	2	8	5,7
38	Chandran	60/m	y	y	y	y	Y	N	N	Diffusely enlarged pancreas	5	4	5,7
39	Vasu	53/m	y	y	y	y	Y	N	N	Diffusely enlarged pancreas	4	6	5,7

40	Arumugam	65/m	y	y	y	y	Y	N	N	Diffusely enlarged pancreas	8	6	5,7
41	Murugan	68/m	y	y	y	y	Y	N	N	Diffusely enlarged pancreas	9	6	5,7
42	Devanathan	59/m	y	y	y	y	Y	N	N	Diffusely enlarged pancreas	2	0	5,7
43	Mohan	55/m	y	y	y	y	Y	N	N	Diffusely enlarged pancreas	11	0	5,7
44	Boopathy	51/m	y	y	y	y	Y	N	N	Diffusely enlarged pancreas	3	4	5,7
45	Manickam	25/m	y	y	y	y	Y	N	N	Diffusely enlarged pancreas	0	4	NIL
46	Kathirvel	60/m	y	y	y	y	Y	N	Fluffy lung infiltrates,left pleural effusion	Diffusely enlarged pancreas	16	8	1,3,4,5,6,7,9,
47	Karthikeyan	40/m	y	y	y	y	Y	n	Fluffy lung infiltrates, left pleural effusion	Diffusely enlarged pancreas	12	8	3,5,6,7,9
48	Narayanan	55/m	y	y	y	y	Y	y	Fluffy lung infiltrates, right pleural effusion	Diffusely enlarged pancreas	15	8	5,6,7,9
49	Sangapillai	47/m	y	y	y	y	Y	y	Fluffy lung infiltrates, left pleural effusion	Diffusely enlarged pancreas	12	8	1,3,5,6,7,9
50	Balamurugan	38/m	y	y	y	y	y	y	normal	Enlarged pancreas	12	4	1

LIST OF COMPLICATIONS AND THEIR KEY WORDS USED IN MASTER CHART

1. ACUTE FLUID COLLECTION
2. VENOUS THROMBOSIS
3. ACUTE RESPIRATORY DISTRESS SYNDROME
4. PANCREATIC ASCITES/ EFFUSION
5. PARALYTIC ILEUS
6. ORGAN FAILURE/ MULTIORGAN DYSFUNCTION SYNDROME
7. PANCREATIC NECROSIS
8. INTESTINAL OBSTRUCTION
9. HYPOCALCEMIA/HYPERGLYCEMIA
10. PSUEDOCYST
11. INTESTINAL PERFORATION
12. DIC/ ENCEPAHLOPATHY